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Cardiac response of trained and untrained older males during upright cycle ergometry

Peter McLaren
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Cardiac Response of Trained and Untrained Older
Males During Upright Cycle Ergometry

A thesis submitted in partial fulfillment of the requirements

for the award of the degree

Honours Master of Science

from

The University of Wollongong

Peter McLaren B.Ed.



Department of Biomedical Sciences
1995

Cardiac Response of Trained and Untrained Older

Males During Upright Cycle Ergometry

Abstract

Cardiovascular responses of trained male Cyclists (65 ± 2.1 years; $n=10$), trained Runners (65 ± 3.4 years; $n=11$), and untrained healthy Controls (66 ± 1.1 years; $n=10$) were examined at supine and seated rest and during incremental upright cycle ergometry ($20 \text{ W} \cdot \text{min}^{-1}$). Mean aerobic power of Cyclists ($53.75 \pm 1.58 \text{ ml} \cdot \text{kg}^{-1} \text{ min}^{-1}$) was significantly higher ($p < 0.05$) than that of Runners ($47.95 \pm 3.94 \text{ ml} \cdot \text{kg}^{-1} \text{ min}^{-1}$), whereas values for both groups were significantly higher than that of Controls ($28.42 \pm 1.26 \text{ ml} \cdot \text{kg}^{-1} \text{ min}^{-1}$). Resting heart rate of Cyclists ($56 \pm 1.26 \text{ b} \cdot \text{min}^{-1}$) and Runners ($51 \pm 2.57 \text{ b} \cdot \text{min}^{-1}$) were significantly lower than that of Controls ($68 \pm 3.43 \text{ b} \cdot \text{min}^{-1}$). Using impedance cardiography, stroke volume and stroke index, cardiac output and cardiac index, and total peripheral resistance response to exercise was compared. Also, blood pressure and rate pressure product were measured throughout exercise. Cyclists' and Runners' stroke volume significantly increased by 41% and 47% at a heart rate of $90 \text{ b} \cdot \text{min}^{-1}$ and continued to rise throughout exercise, whereas stroke volume of the Control subjects increased by 31%. Cardiac index and stroke index of Runners and Cyclists was significantly higher than that of Controls during exercise. In addition, Runners' total peripheral resistance and rate pressure product response were significantly lower than Cyclists and Controls throughout exercise. These data indicate that trained older Cyclists, Runners, and untrained Controls were able to significantly increase stroke volume and decrease total peripheral resistance during exercise. During exercise, however, Cyclists and Runners displayed greater stroke volume than Controls which was one

of the major contributing factors for their greater $\dot{V}O_{2peak}$. Also, stroke volume peaked at less than 20% of $\dot{V}O_{2peak}$ in both the trained and untrained older subjects. This early increase in stroke volume was a determining factor of the increased cardiac output during exercise not only in the trained but also in the untrained, healthy Controls. Overall the Cyclists displayed the greatest $\dot{V}O_{2peak}$ although the Runners had a larger stroke volume during exercise. The finding that Runners recorded both a large increase in stroke volume and a high $\dot{V}O_{2peak}$ suggests that running results in enhanced stroke volume response to cycle ergometry in older males.

Dedication

To Lexie for all your support.

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CHAPTER ONE: INTRODUCTION

1.1 RATIONALE

The elderly proportion of the Australian population is continuing to expand. For example, during the last two decades the number of Australians aged over 65 years has grown by 89% (Australian Bureau of Statistics, 1994). Also, it has been predicted that by 2030 the elderly will be the fastest growing segment of the population and the number of people aged 80 years or more will have increased by over 1 million (Australian Bureau of Statistics, 1993). The health implications of this expansion are enormous because the majority of the Australian elderly population currently suffers from a variety of chronic diseases, with cardiovascular disease and reduced cardiac function being especially prevalent (Heart Facts, 1991). Thus the incidence of cardiovascular disease and its resultant debilitation of cardiac performance are expected to increase in the future (Olshansky *et al.*, 1993; Pendergast *et al.*, 1993).

Although the age-associated decline in cardiovascular function is inevitable other factors such as diet and sedentary lifestyle contribute to the degradation in cardiac performance. The latter is particularly important, because aerobic exercise appears to negate or at least reduce the typical decline in cardiac function of the older population. Thus, endurance-trained older males have significantly greater aerobic power ($\dot{V}O_{2peak}$) compared to that of untrained age-matched older males (Heath *et al.*, 1981; Ogawa, *et al.*, 1992., Pollock *et al.*, 1987; Rogers *et al.*, 1990).

Previous research on cardiovascular adaptations to endurance training has primarily focused on young subjects. For example, increased aerobic power which

is mediated by enhanced oxygen extraction and cardiac output. Enhanced cardiac output has been attributed to an enlarged stroke volume because maximal heart rate is not increased through training (Milliken *et al.*, 1988; Rerych *et al.*, 1980; Spina *et al.*, 1992). Because maximal heart rate declines with age (Lakatta, 1993), regardless of training status, any elevation in cardiac output brought about through training adaptation must occur via an enlargement in stroke volume. Stroke volume typically declines with age but is increased with regular physical activity (Lakatta, 1993). Recent research indicates that the basis of trained older males increased aerobic power is their superior ability to increase stroke volume during exercise, which, in turn, allows them to significantly increase cardiac output (Fleg *et al.*, 1994a; Seals *et al.*, 1994). Thus, similar to the trained young, aerobic power in trained older subjects is significantly influenced by stroke volume (Fleg *et al.*, 1994a; Hagberg *et al.*, 1985; Ogawa *et al.*, 1992; Seals *et al.*, 1994)

However, actual stroke volume dynamics during light to heavy incremental exercise are unclear in both young and older subjects. For instance, the increased stroke volume response to progressive upright exercise is generally thought to be a linear increase to a maximal value, followed by a levelling off at approximately 40-50% of $\dot{V}O_{2max}$ (Astrand *et al.*, 1964; Higgenbotham *et al.*, 1986; Poliner *et al.*, 1980; Sullivan *et al.*, 1991). Whether or not stroke volume peaks at lower levels of $\dot{V}O_{2max}$, however, is undetermined mainly because of measurement limitations. For instance, traditional methods of measuring stroke volume are either invasive or require subjects to be exercising at steady state. Thus, although the stroke volume response to light exercise in the trained is unclear past research has consistently shown that young sedentary subjects fail to further increase stroke volume once

exercise progresses from light into moderate and heavy exercise (Schairer *et al.*, 1992; Spina *et al.*, 1992; Tanaka *et al.*, 1986). In contrast, the stroke volume response to exercise in untrained older males has shown quite diverse patterns, either increasing (Rodheffer *et al.*, 1984), staying the same (Strandell, 1964), or decreasing (Brandfonbrener *et al.*, 1955). Thus, the stroke volume response to incremental aerobic exercise of the untrained older individual remains undetermined.

Thus, the main purpose of this study was to compare the stroke volume response dynamics of trained older Cyclists and healthy untrained Controls during rest and upright cycle ergometry exercise.

1.2 SIGNIFICANCE

Given that the proportion of the Australian population who are over 65 years is expanding, knowledge of physiological changes that are detrimental to quality of life are becoming increasingly more important. Because older individuals are characterised by reduced cardiovascular function it is important to determine the basis of the enhanced cardiovascular dynamics of the endurance trained older person.

Trained older subjects should display enhanced functional capacity, characterised by a larger cardiac output and an increased stroke volume. Because of improved myocardial efficiency an enhanced exercising stroke volume has major health implications. For example, an enhanced exercising stroke volume response better facilitates oxygen transport, thereby minimising the physiological impact of both daily and unexpected stress. Similarly, there are potential health benefits if untrained, older asymptomatic males who are healthy and active were to also demonstrate enhanced cardiac function during exercise. If this study demonstrates

that regular aerobic exercise is associated with improved cardiac function, then aerobic exercise will be further supported as a potential health intervention to preserve physical capacity during the ageing process.

Previous research with young males has also shown that high intensity running results in increased $\dot{V}O_{2peak}$ during cycle ergometry (Boutcher *et al.*, 1989). However, whether trained older Runners also possess enhanced $\dot{V}O_{2peak}$ during cycle ergometry has not been established. If trained older Runners are able to record enhanced $\dot{V}O_{2peak}$ during cycle ergometry, similar to their trained cycle counterparts, they should also demonstrate elevated exercise stroke volume. There is a paucity of research investigating the specific role of specific modes of exercise and cardiac adaptations with older males. That is, the enhancement of performance and cardiac function at one exercise modality, through adaptation to a second form of exercise. Thus, it is feasible that trained older runners may also demonstrate an enhanced stroke volume response during cycling. Also, older runners participating in cross-training such as cycling may accrue additional benefits. For instance, older runners would be at least similarly predisposed, if not more so, to injury than young runners, who typically have a high injury rate (Mutton *et al.*, 1993). Thus, older runners, training on the bicycle, may increase training volume without elevating injury risks, may improve training variety, and may also accrue cardiac adaptations, such as increased stroke volume by participating in cycle training. Thus, a second aim of the study was to compare the stroke volume response of trained older Runners to that of age-matched Cyclists during upright cycle ergometry.

1.3 AIMS

The specific aims of this study were to:

- i) determine untrained older males stroke volume response to exercise.
- ii) compare stroke volume response at rest and during incremental cycle ergometry exercise of trained older male Cyclists to that of untrained Controls.
- iii) determine at what percentage of $\dot{V}O_{2peak}$ stroke volume peaks during incremental exercise for trained and untrained older males.
- iv) compare the stroke volume response of trained older Runners to that of age-matched Cyclists during incremental cycle ergometry.

1.4 HYPOTHESES

It is hypothesised that, compared to untrained older controls, the trained older Cyclists and older Runners will display:

- i) significantly lower heart rate, total peripheral resistance, blood pressure, and greater stroke volume during rest.
- ii) significantly greater cardiac output, cardiac index, stroke volume, stroke index, and lower heart rate, total peripheral resistance, and blood pressure at each absolute work load during incremental exercise.
- iii) finally it is hypothesised that during cycle ergometry trained older Runners will display similar stroke response to that of trained older Cyclists.

CHAPTER TWO: LITERATURE REVIEW

The initial focus of the literature review will be to describe the age-related cardiovascular changes and the factors that determine cardiac performance. The impact of aerobic exercise upon elderly cardiac function will also be examined together with the factors underlying the greater cardiovascular efficiency of the trained older individual. In addition, the review will focus upon the methodology employed to measure cardiac output during exercise, with particular emphasis upon impedance cardiography, which was the indirect method of determining stroke volume in this study. Finally, the review will examine previous research describing stroke volume response dynamics to exercise, modes of testing and posture, cardiovascular health of the population, and the subjects' specific modality of training.

2.1 AGEING AND CARDIOVASCULAR STRUCTURE AND FUNCTION

Cardiovascular function declines with age, however, this inevitable decline may not necessarily be a consequence of ageing but also may be influenced by disease, sedentary living, and deconditioning which is more prevalent among the older populations. Typical findings of age-associated changes in the myocardium and vasculature that have important consequences for cardiovascular performance include morphological and functional changes. For example, increased collagen content, amyloid deposition, fibrosis of heart and vasculature, decreased myocardial and arterial compliance, calcification of the left side of the cardiac skeleton, fat accumulation around the sinoatrial node together with a decreased number of pacemaker cells, decreased sensitivity of the sinoatrial node to catecholamines, and a diminished capacity of the autonomic reflexes controlling blood flow are all

associated with ageing (Feldman, 1986; Hutchins, 1980; Lakatta *et al.*, 1975; Lakatta, 1993; Miller *et al.*, 1986; Nixon *et al.*, 1985; Wei, 1992; Wei *et al.*, 1984). These morphological changes are associated with functional degradation that include decreased maximum heart rate, stroke volume, and cardiac output (Fleg, 1986; Lakatta, 1993). These changes result in a dramatic decrease in cardiovascular efficiency and may limit aerobic power (Dehn & Bruce, 1972; Heath *et al.*, 1981; Rivera & Thompson, 1989). As a result, one of the most consistent findings in gerontological research is the age-related decline in maximal oxygen consumption ($\dot{V}O_{2max}$) which averages between 5% and 10% per decade between the ages of 25 and 75 years (Dehn & Bruce, 1972; Raven & Mitchell, 1980). Also, ventricular relaxation and contraction are prolonged (Douglas *et al.*, 1992; Lakatta *et al.*, 1975) and blood pressure is increased due to elevated peripheral resistance to blood flow (Fleg, 1986; Julius *et al.*, 1967; Lakatta, 1993). Collectively, these results indicate that the morphological changes associated with ageing appear to result in a decline in cardiac performance that may impact upon physical performance. Thus, when older sedentary individuals are physiologically stressed, they experience greater physiological strain, and their capacity to perform work is reduced relative to that of younger individuals.

2.2 FACTORS INFLUENCING CARDIAC PERFORMANCE

Systemic cardiac output, the product of heart rate and stroke volume, is usually defined as the volume of blood ejected from the left ventricle per minute. To ensure respiratory gas exchange, metabolic removal and blood pressure maintenance, cardiac output adjusts in accordance with the metabolic requirements of the body (Guyton, 1990). Because resting cardiac output must deliver blood to all cells, the

resting cardiac output is strongly correlated with body mass and surface area. For example, average cardiac output of a healthy adult at rest is approximately 3.2 L.min⁻¹ of body surface area. Therefore, cardiac output of a 70 kg adult would be 5.6 L.min⁻¹ (Smith & Kampine, 1990). Also, cardiac output is influenced by age. Cross-sectional studies indicate that with advancing age cardiac output tends to decrease (Branfonbrener *et al.*, 1955; Julius *et al.*, 1967; Ogawa *et al.*, 1992; Strandell, 1964). In addition, maximal heart rate and stroke volume typically decline with age (Lakatta, 1993). Also the frequency of the cardiac cycle is a major determinant of cardiac output. Usually, up to a ceiling point, the higher the frequency the more blood the heart can pump, however, there are limitations. For instance, the increase in cardiac output with increased oxygen uptake becomes smaller at higher heart rates, due to decreased diastolic filling time (Astrand, 1964), which counters the cardiac output rise by lowering stroke volume.

Heart rate is determined by the rate of impulse generation in the sinoatrial node, which, in turn, is determined by the balance between parasympathetic and sympathetic stimulation. For example, at the onset of exercise and during light exercise, a decrease in parasympathetic impulses to the sinoatrial node is the main mechanism for increasing heart rate (Seals *et al.*, 1994). Heart rate is also influenced by cardiovascular reflexes. For example, at the initiation of dynamic large muscle exercise, if the reduction of parasympathetic impulses fails to elevate heart rate and cardiac output sufficiently to match vascular conductance, then increased sympathetic activity enables heart rate to increase cardiac output and blood pressure (Rowell & O'Leary, 1990).

Systemic stroke volume is the amount of blood pumped into the aorta with

each cardiac contraction. This volume is almost maximal in the supine position (it only increases slightly during supine exercise), and decreases by about one third in the upright position, due to changes in blood volume distribution and venous pooling, and their influence on venous return to the right atrium (Saltin, 1969). Stroke volume is the major contributor to cardiac output in the transition from rest to moderate upright exercise (Astrand *et al.*, 1964). Unlike heart rate, which increases linearly with work rate and $\dot{V}O_{2max}$, stroke volume is generally believed to reach a maximal value, at approximately 40% of $\dot{V}O_{2max}$, plateauing thereafter (Astrand *et al.*, 1964).

2.3 FACTORS DETERMINING STROKE VOLUME

Stroke volume is determined by many inter-dependent factors which include preload, inotropic and chronotropic states, afterload, and distensibility of the ventricles (Lakatta, 1993). For example, myocardial contractility is directly influenced by inotropic state, whereas afterload and distensibility exert mainly mechanical effects on stroke volume. In addition, each of these inter-dependent factors are controlled by autonomic modulation, which forms the basis of several cardiovascular reflexes (Lakatta, 1993). The following description briefly summarises these inter-relationships.

Preload refers to the amount of blood filling in the ventricles via the atria (venous return) during diastole (Lakatta, 1993). Increased preload causes greater cardiac filling which, increases end-diastolic volume, stretching (preloading) the myocardial fibers, improving contractility, and increasing stroke volume (the Frank-Starling mechanism: Green & Crouse, 1991). The Frank-Starling mechanism, and left ventricular end-diastolic volume, are directly affected by body position

(maximum venous return when supine), the pumping action of respiration, skeletal muscle pump, total blood volume, and cardiac output (Rowell, 1986). For example, as a result of prolonged, standing at attention the muscle pump no longer facilitates the return of blood to the heart, consequently stroke volume decreases.

Ventricular distensibility (compliance) refers to the capacity of ventricles to stretch and accommodate diastolic filling. During ageing, the deposition of collagen into the myocardium (Hutchins, 1980) results in the ventricles becoming less compliant, and consequently limiting stroke volume increases during exercise because of a reduced ability to accommodate the increased venous return that occurs during exercise (Folkow & Svanborg, 1993). Similarly, ageing influences afterload (mechanical impedance) encountered subsequent to ventricular contraction (Lakatta, 1993). Age-related atherosclerosis and stenoses increase resistance to ventricular emptying, negatively affecting stroke volume, and elevating cardiac work. For example, in hypertension, high peripheral resistance to blood flow results in an increased afterload stress that results in a lowered stroke volume (Rowell, 1986).

Inotropic state refers to the strength of contraction of the heart, and at constant conditions of loading and heart rate the quality of ventricular performance (Guyton, 1990). The strength of myocardial fibre shortening is significantly increased through the stimulation of sympathetic nerves releasing catecholamines (Folkow & Svanborg, 1993; Seals *et al.*, 1994) and occurs independently of end-diastolic volume and afterload (Lakatta, 1976). Therefore, at any given level of venous return and afterload, increased myocardial contractility will result in a larger stroke volume and lower end-systolic volume.

Overall, stroke volume is determined by the interaction of many

interdependent factors. Based on these interactions any factor which increases end-diastolic volume and decreases end-systolic volume will result in an increased stroke volume. Conversely, stroke volume will be lowered by factors that decrease end-diastolic volume and increase end-systolic volume. Many of these factors are associated with both the ageing process and hypokinesia.

2.4 AGEING AND AEROBIC EXERCISE

Although decreases in cardiovascular function accompany ageing, other factors, such as heart disease and body composition (Toth *et al.*, 1994) contribute to the degradation in cardiac performance in the elderly (Lakatta, 1993., Wei, 1992). Also many of the cardiovascular changes associated with ageing are similar to those accompanying habitual inactivity (Stratton *et al.*, 1994). The influence of sedentary living is especially important, as habitual endurance exercise appears to be able to offset the typical decline in the cardiac function of the older individual (Rogers *et al.*, 1990).

Because cardiac function is one of the determinants of aerobic power, it is possible that age-related decrements in peak cardiac output may result in a decline in aerobic power with age. In fact this is a fairly universal observation. In particular, cardiac output, stroke volume, and $\dot{V}O_{2\text{peak}}$ are severely compromised. The extent of the decline in aerobic power with age fluctuates according to variations in body mass, population sampling, and existing fitness level of subjects (Lakatta, 1993). Numerous studies indicate that regular exercise among elderly individuals at the appropriate intensity, duration, and frequency can increase maximal oxygen uptake level (Blumenthal *et al.*, 1991; Cononie, 1991; Ehsani *et al.*, 1991; Seals *et al.*, 1984., Shocken *et al.*, 1983). Thus, the rate of decline in $\dot{V}O_{2\text{max}}$ is much greater in

sedentary than in active older men (Dehn & Bruce, 1972). Both longitudinal (Dehn & Bruce, 1972; Hagberg *et al.*, 1981; Rogers *et al.*, 1990) and cross-sectional studies indicate that elderly individuals who regularly participate in endurance exercise, typically have up to twice the $\dot{V}O_{2\max}$ of their sedentary counterparts (Fleg *et al.*, 1994a; Hagberg *et al.*, 1985; Ogawa *et al.*, 1992., Pollock *et al.*, 1987; Seals *et al.*, 1994). Furthermore, elite master runners (age ranging from 50 to 72 years) have been shown to possess greater $\dot{V}O_{2\max}$ than that of young untrained males (Heath *et al.*, 1981; Figure 1). Consequently, it is reasonable to assume that older endurance-trained athletes may have enhanced cardiovascular function contributing to their increased aerobic power.

In much the same manner as in young adults, the cardiovascular system of older subjects shows clear signs of endurance training adaptation. Recently, Kohort *et al.* (1991) and Makrides *et al.* (1990) observed that gains in aerobic power of older subjects are of the same magnitude as that observed in young adults. Also these increases in aerobic power are associated with increases in vascular conductance, maximal cardiac output, and stroke volume. Similarly, investigations comparing older endurance athletes with young athletes demonstrate that the training-induced decrease in resting heart rate also occurs in the older population (Hagberg *et al.*, 1985; Heath *et al.*, 1981; Seals *et al.*, 1994). In addition, heart rate responses to submaximal exercise are reduced by endurance training (Seals *et al.*, 1984). Aerobic training of older individuals does not appear to affect resting systolic blood pressure (Meredith *et al.*, 1989), or diastolic blood pressure (Sagiv *et al.*, 1989; Seals *et al.*, 1984). Recently, physical conditioning has been associated with a reduction in the age-associated diastolic dysfunction (Douglas *et al.*, 1992).

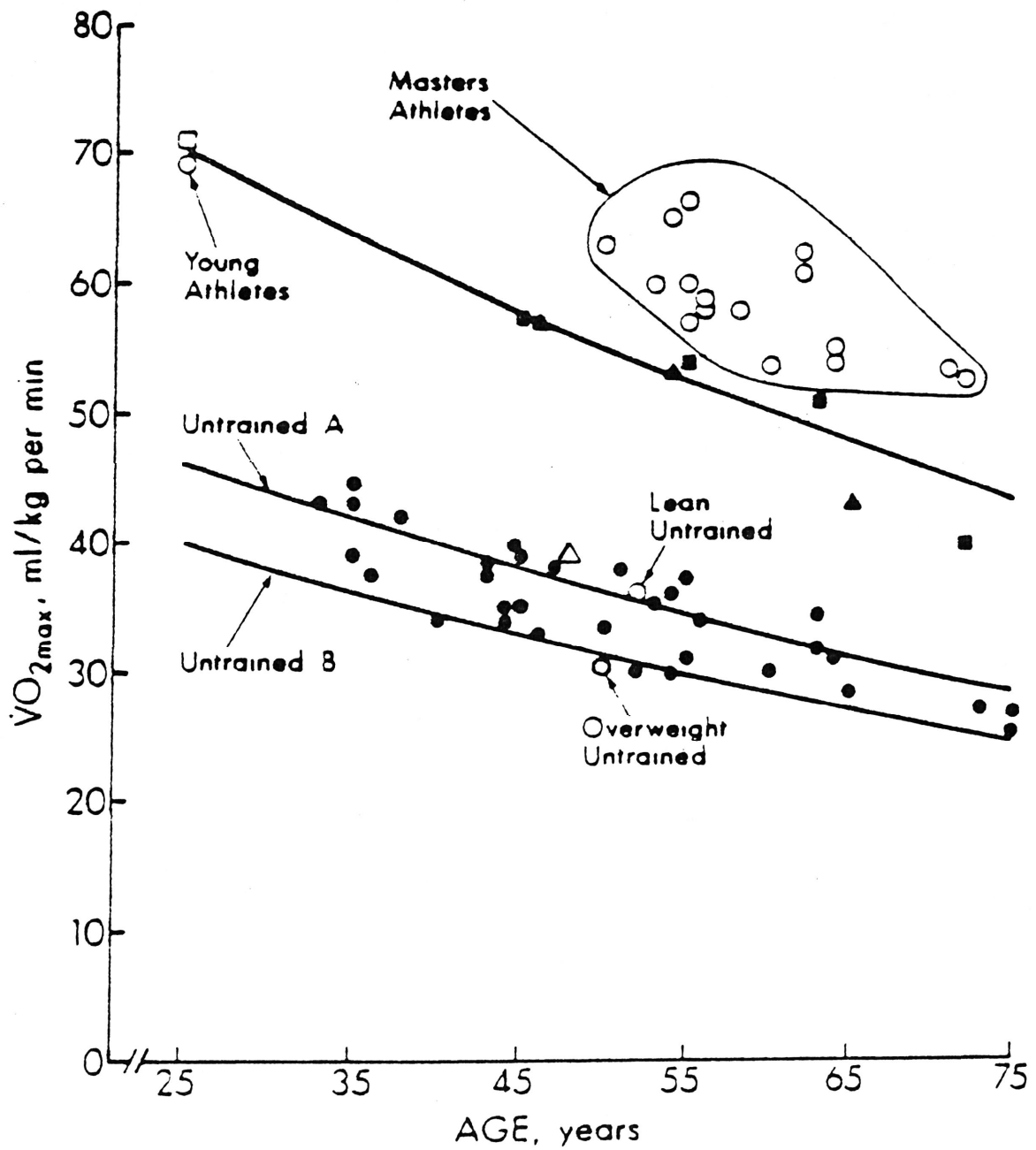


Figure 2.1 Rate of decline in $\dot{V}O_{2max}$ for young and older lean and obese untrained, together with young and masters athletes (From Heath *et al.*, 1981).

Also, during submaximal exercise, trained older athletes compared to untrained age-matched controls display lower blood pressure and systemic vascular resistance (Hagberg *et al.*, 1985; Heath *et al.*, 1981; Seals *et al.*, 1984). Also, compared to untrained, trained older males display lower blood pressure and vascular resistance during exercise (Martin *et al.*, 1991). Of critical importance to the current project is the training-induced elevation in stroke volume.

One of the major factors underlying the high aerobic power observed in elite young athletes is their ability to increase stroke volume during exercise (Rerych *et al.*, 1988). Saltin (1969) found that a group of highly trained male athletes possessed a mean maximal stroke volume of 160 ml compared to a maximal stroke volume of 100 ml for a group of sedentary males. Resting values for the athletes versus the sedentary were 100 ml and 80 ml respectively. Thus, dramatic differences in resting and exercise stroke volume exist between trained and untrained young individuals.

Enhanced stroke volume has recently been identified, as a major cardiac adaptation underlying enhanced cardiac output of older males during upright cycle ergometry. Fleg and colleagues (1994a), using radionuclide ventriculography, attributed the significantly greater cardiac index of trained older athletes (62.8 ± 6.9 years, 50.1 ± 4.9 ml.kg⁻¹min⁻¹) during maximal upright cycle exercise to an augmented stroke volume index. Similarly, Seals and colleagues (1994), using echocardiography, demonstrated enhanced stroke volume to be the major cardiac adaptation underlying the improved cardiac output of the masters athletes (64 ± 2 years, 50.4 ± 1.7 ml.kg⁻¹min⁻¹) compared to sedentary controls (63 ± 1 year, 29.6 ± 1.4 ml⁻¹.kg⁻¹min⁻¹). However, subjects in this study were tested during supine

exercise. Also, during supine cycling, Ehsani *et al.* (1991) demonstrated that previously sedentary, men who underwent twelve months of intense endurance training, significantly increased end-diastolic volume and stroke volume at maximal exercise. Thus, stroke volume augmentation is a major cardiovascular adaptation of older endurance trained athletes during both supine and upright exercise.

Hagberg *et al.* (1985) have shown that elite older runners possessing high $\dot{V}O_{2\max}$ have both greater stroke volume and increased arterio-venous oxygen difference during treadmill exercise. These authors suggest that the larger stroke volume and greater arterio-venous oxygen difference contribute equally to the enhanced $\dot{V}O_{2\max}$ of older runners. The contribution of an increased arterio-venous oxygen difference of the trained older athlete is primarily caused by cellular adaptations in the exercising muscles (Coggan *et al.*, 1993; Suominen *et al.*, 1977a; Suominen *et al.*, 1977b). However, the mechanisms underlying the increased stroke volume of trained older males have not been identified.

2.5 MECHANISMS UNDERLYING INCREASED STROKE VOLUME

Schairer *et al.* (1992) have demonstrated that the large exercise stroke volume of elite young cyclists and runners was influenced primarily by increased preload (which is reflected in end-diastolic volume) and to a lesser extent by myocardial contractility. This indicates that, in younger subjects, endurance training may increase cardiac performance by inducing cardiac dilatation during exercise. Such dilatation may also presumably be present during rest, as young endurance-trained athletes typically possess significantly larger resting stroke volumes, and lower resting heart rates compared to sedentary individuals (Schairer *et al.*, 1991). This is supported by Tanaka *et al.* (1986) and Rerych *et al.* (1980), who found that

prolonged training led to a greater left ventricular volume (due to a facilitation of left ventricular filling), to an increase in systolic emptying (possibly related to a reduction in total peripheral resistance), and to a greater myocardial contractility compared to untrained individuals. Thus, in young subjects, the increase in end-diastolic volume was influenced by venous return and reduced myocardial stiffness (Levine *et al.*, 1991; Rerych *et al.*, 1980), whereas the decrease in end-systolic volume is determined by increased myocardial contractility (Sagiv *et al.*, 1989).

Similarly, the main factors underlying the increased stroke volume of the trained elderly are increased end-diastolic volume (Seals *et al.*, 1994; Stratton *et al.*, 1994) and decreased end-systolic volume (Ehsani *et al.*, 1991; Stratton *et al.*, 1994). However, their precise roles and underlying mechanisms are still unclear. An investigation by Seals *et al.* (1994) attempted to separate the mechanisms, determining the enhanced stroke volume and cardiac output in master athletes. Increased stroke volume from rest to exercise was positively correlated with changes in end-diastolic volume in both the master athletes ($r=0.78$) and sedentary controls ($r=0.73$). During rest and exercise, systolic and mean blood pressure did not differ between the two groups. However, end-systolic volume of the trained elderly decreased significantly during exercise. The trained subjects also had a larger increase in stroke volume for any given increase of end-diastolic volume, indicating that enhanced systolic function and a greater preload acted as independent mechanisms resulting in improved cardiac function in the trained elderly subjects. Similarly, augmented stroke volume and left ventricular contractility have been observed in previously sedentary elderly males subsequent to endurance cycle training (Ehsani *et al.*, 1991; Stratton *et al.*, 1994). Thus, older trained subjects

appear to have enhanced venous return (end-diastolic volume), decreased end-systolic volume, and an increased stroke volume at rest and during exercise.

Young endurance athletes have been shown to have more compliant and distensible ventricles (Levine *et al.*, 1993). Also, the ability of ventricular compliance to enhance stroke volume has been demonstrated in pericardiectomised dogs (Stray-Gundersen *et al.*, 1986). These dogs increased stroke volume by 25% which enabled cardiac output to be significantly elevated. Thus, it is feasible that aerobic training may alter the compliance of the myocardium, which in turn, will accommodate a larger end-diastolic volume, though there have been very few projects which have tested this hypothesis (Convertino, 1991). However, the ability of aerobic exercise to increase myocardial contractility cannot be discounted. As mentioned earlier, the increased stroke volume of the trained elderly may be as a result of a more powerful heart ejecting more blood from the ventricles (Ehsani *et al.*, 1991; Sagiv *et al.*, 1989). Previous research with rats indicates aerobic exercise increases myocardial contractility and reduces the stiffness of the myocardium (Spurgeon *et al.*, 1983) although its importance in the human heart remains unclear.

If increased end-diastolic volume is responsible for the enhanced stroke volume response of the endurance-trained elderly, then increased venous return and cardiac compliance would appear to be prime underlying adaptations. Indirect support for an enhanced venous return comes from blood-volume manipulation studies, which have shown that hypervolaemia results in an elevation in cardiac output and stroke volume (Hopper *et al.*, 1988; Kanstrup *et al.*, 1982). Total blood volume is an important factor in the regulation of circulatory function, which in healthy adults does not appear to change with age (Strandell, 1964). Also, blood

volume is transiently increased after training in both young and old subjects (Benstead, 1965; Carroll *et al.*, 1995; Panton *et al.*, 1994), and this enhanced blood volume appears to increase the ability of the trained to maintain a higher stroke volume with a lower heart rate during exercise (Convertino, 1991). Conversely, Schulman *et al.* (1992) reported that as a result of twelve weeks of deconditioning, older athletes experienced an 18% decrease in peak cardiac index due to a reduction of stroke volume index from 66 ml/m² to 54 ml/m². Similarly, Coyle *et al.* (1986) concluded that, as a result of a two weeks of detraining, stroke volume declined by 12%, due to a 9% decrease in blood volume which appeared to limit ventricular filling during exercise. Thus, enhanced blood volume may be an adaptation underlying the enhanced stroke volume of endurance trained older athletes.

2.6 STROKE VOLUME RESPONSE TO EXERCISE

To this point, the factors which regulate stroke volume have been explored, but the dynamics of the stroke volume response to exercise have not been discussed. Previous research indicates that stroke volume first increases then plateaux. However, there is no universal agreement on the relative exercise intensity at which this plateau occurs. For example, Brooks and Fahey (page 335., 1990) report stroke volume to plateau at approximately 25% of $\dot{V}O_{2max}$. In contrast, it has been suggested that stroke volume plateaux at approximately 40-50% of $\dot{V}O_{2max}$, which corresponds to a heart rate of approximately 110-120 b.min⁻¹ (Astrand *et al.*, 1964; Sullivan *et al.*, 1991). Sullivan and colleagues (1991) examined stroke volume response using right-sided cardiac catheterisation and radionuclide angiography on normal males and females (aged 20 to 70 years) and found that stroke volume peaked at 50% peak oxygen consumption and remained unchanged during intense

exercise. These findings are in agreement with other studies (Higginbotham *et al.*, 1986; Poliner *et al.*, 1980; Saltin, 1969). These apparent discrepancies in the temporal response of stroke volume to exercise may be attributable to methodological differences, training status, and the age of subjects. One of the purposes of the present investigation is to examine the dynamics of the stroke volume response to incremental exercise in trained and untrained older males.

Schairer *et al.* (1992) have previously demonstrated that a larger stroke volume response underlies the greater cardiac output of the trained young males compared to sedentary controls. Schairer and colleagues obtained echocardiographs of subjects during bicycle ergometry (semi-upright body position) at three stages of exercise. Each stage was 3 minutes in duration at heart rates of 110, 130, and 150 b.min⁻¹. The athletes' stroke volume increased prominently from rest to exercise at the first data collection point (i.e., heart rate of 110 b.min⁻¹) and appeared to peak at this heart rate. In contrast, at the same heart rate, the sedentary subjects had no significant increase in stroke volume. Similarly, the findings of Levine *et al.* (1991) indicate that the sedentary individual in the transition from rest to exercise possesses a small rate of increase in stroke volume compared to the trained individual who has a considerably larger increase. This is because the sedentary individual relies predominately on heart rate to increase cardiac output, whereas the trained individual uses both heart rate and stroke volume to increase cardiac output (Stein *et al.*, 1980). Crawford *et al.* (1985), using two-dimensional echocardiography, and Goodman *et al.* (1991), using radionuclide angiography, observed increases in stroke volume and end-diastolic volume in both trained and untrained groups, with the trained subjects showing greater increases in end-diastolic volume throughout

exercise. Thus, young trained athletes, compared to sedentary controls, display a larger stroke volume response to exercise.

It has also been demonstrated that the young trained individual reaches maximal stroke volume at a higher relative $\dot{V}O_{2\max}$ (Tanaka *et al.*, 1986) and that the trained individual will have a greater percent increase in stroke volume than the untrained (Barnard *et al.*, 1980; Levine *et al.*, 1991). Tanaka *et al.* (1986) using impedance cardiography demonstrated that young trained subjects' stroke volume plateaued or slightly increased from heart rates of 120 b.min⁻¹ to maximal heart rate, whereas untrained subjects experienced a reduction in stroke volume at heart rates above 130 b.min⁻¹. Similarly, Spina *et al.* (1992) reported that young untrained subjects decreased their stroke volume between 50 and 100% of $\dot{V}O_{2\max}$ and after a training intervention increased their stroke volume between 50 and 100% of $\dot{V}O_{2\max}$. Also, Crawford *et al.* (1985), examined stroke volume and measures of left ventricular function in competitive and non-competitive runners at rest and during upright exercise (60, 70, and 85% of maximal heart rate) on a cycle ergometer using two-dimensional echocardiography. Stroke volume increased significantly in the competitive group through to 70% of maximal heart rate. These findings are supported by recent data from Gledhill *et al.* (1994) which indicated that young athletes continued to increase stroke volume right through to maximal exercise. Thus, trained young athletes typically increase stroke volume during submaximal exercise, whereas young sedentary subjects typically reduce stroke volume during submaximal exercise.

Data from our laboratory (Boutcher *et al.*, 1994), using impedance cardiography, indicated that stroke volume of elite young cyclists peaked at less than

20% of $\dot{V}O_{2\max}$. The pattern of stroke volume response exhibited by the trained was characterised by a large stroke volume increase early on during exercise that did not reduce with higher exercise heart rates. The plateauing of stroke volume at a heart rate of 90 b.min⁻¹ (less than 20% of $\dot{V}O_{2\max}$) suggests that the largest increase in stroke volume for the trained occurred early on in exercise. Similarly, in fit but untrained subjects, stroke volume was already at near maximal levels during the first collection point, which occurred at a heart rate of 90 b.min⁻¹. However, in contrast to the trained cyclists their stroke volume was significantly lower during exercise. These results support prior research (Schairer *et al.*, 1992) that has shown that young aerobically trained athletes possess a greater stroke volume during exercise compared to untrained subjects. These data may imply that stroke volume during exercise peaks at a lower percentage of $\dot{V}O_{2\max}$ than previously thought.

There is a paucity of research describing stroke volume response dynamics of older subjects to exercise. However, previous research with young subjects indicates that individuals with high $\dot{V}O_{2\max}$ tend to also possess a higher stroke volume. Similarly, if trained older athletes have larger blood volume and venous return then it is feasible that they will also display similar stroke volume dynamics to that of the young. However, the stroke volume response dynamics of trained older athletes and untrained older males during the early phase of exercise is unresolved.

Although the stroke volume response to light exercise in the trained is unclear past research, as previously described, has consistently shown that young sedentary subjects fail to increase stroke volume during moderate to heavy exercise (Schairer *et al.*, 1992; Spina *et al.*, 1992; Tanaka *et al.*, 1986). In contrast, the

stroke volume response to exercise in untrained older males has been shown to either increase (Rodheffer *et al.*, 1984; Younis *et al.*, 1990), stay the same (Strandell, 1964), or decrease (Brandfonbrener *et al.*, 1955; Granath *et al.*, 1964; Julius *et al.*, 1967). The latter investigations were early invasive studies. For example, Granath and colleagues determined stroke volume by the direct Fick method with right heart catheterization. Blood samples were taken at rest and during two separate workloads, approximately 5 minutes into each stage. As a result stroke volume increased in the transition from rest to exercise, however, there was no significant increase during submaximal exercise, (i.e., between the first and second workload). More recently, Ogawa *et al.* (1992) using acetylene rebreathing reported that the stroke volume of untrained older males (63 ± 3 years, 27.2 ± 5.1 ml⁻¹.kg⁻¹.min⁻¹) decreased between 50% and 100% of maximal effort. In contrast, other studies by Rodeheffer *et al.* (1984) and Younis *et al.* (1990), who screened older subjects with exercise ECG and thallium scanning, demonstrated that stroke volume increased during submaximal exercise. Thus, in contrast to young sedentary subjects the stroke volume response during moderate exercise of the untrained older individual is unclear.

2.7 SPECIFICITY OF CARDIAC ADAPTATIONS

A second aim of the study will be to investigate the cardiac response of trained older Runners compared to Cyclists during incremental upright cycle ergometry. If increased blood volume or preload is mediating the increased stroke volume of trained older males then it is feasible that the trained older Runners like trained Cyclists will also demonstrate elevated stroke volume and $\dot{V}O_{2peak}$ during cycle ergometry.

High intensity running and cycle training with young males have been shown to result in similar increases in $\dot{V}O_{2\max}$ (Boutcher *et al.*, 1989; Mutton *et al.*, 1993). Also, similar cardiac adaptations occur in young subjects as a result of endurance training irrespective of the mode of exercise (Wilmore *et al.*, 1980). For instance, McArdle *et al.* (1976) hypothesised that the central circulatory overload caused by various large muscle exercise may have caused the observed reduction in maximum heart rate. This training adaptation may demonstrate an increased oxygen pulse, enhanced oxygen extraction, and/or stroke volume (McArdle *et al.*, 1976). More direct evidence of stroke volume adaptations accruing through cross-training have been supported by the findings of Loftin *et al.* (1988) who demonstrated that increased stroke volume is a major factor determining the increased $\dot{V}O_{2\max}$ of young subjects during peak leg exercise following arm training. Also, Thompson *et al.* (1981) demonstrated adaptations in cardiac function during exercise with untrained limbs. For instance, after training, during submaximal exercise with both trained and untrained limbs, left ventricular ejection time, which correlates positively with stroke volume, was significantly greater (Weissler *et al.*, 1961). Thus, improved exercise performance with untrained limbs provides additional support for cardiac adaptations such as stroke volume, accruing through endurance cross-training. Although previous research has not focused on cross-training and cardiac adaptations with older males, cardiovascular changes with cross-training have been previously identified in middle-aged males (Kasch *et al.*, 1973). Kasch and colleagues prescribed run training over a two-year period to previously sedentary males (mean age of 49 years) and subsequently tested subjects on a bicycle ergometer. As a result $\dot{V}O_{2\max}$ increased by 17% and oxygen pulse ($\dot{V}O_{2\max}/\text{max heart rate}$) increased

by 21%. Consequently it is reasonable to expect male runners over 60 years of age, who as previously mentioned expand blood volume after endurance training, will also exhibit enhanced stroke volume during cycle ergometry.

2.8 CARDIAC OUTPUT MEASUREMENT

The uncertainty surrounding stroke volume response during progressive exercise may be attributed to methodological differences in the measurement of stroke volume measurement. For example, the thermodilution technique involves the insertion of a flow-directed pulmonary artery catheter (Bernstein *et al.*, 1986). This direct technique is highly invasive, and its use is limited during exercise, particularly with the elderly. The potential risks are highlighted by Granath *et al.* (1964) in his description of the complications arising in a circulation study of older men. For instance, during catheterisation the catheter slipped down into the right ventricle and a series of ventricular ectopic beats followed. Also patients fainted and auricular fibrillation occurred when the catheter was in the right atrium. Echocardiography is an indirect technique widely used in the assessment of left ventricular function. It is also limited for exercise researching, as it necessitates subjects to be in the resting supine position for data collection. Carbon dioxide rebreathing is another popular indirect method of estimating stroke volume. However, it is limited by restricted sampling periods and duration, breath-holding requirements (evoking cardiovascular changes), and the necessity of subjects having to reach steady state exercise for 4-5 minutes before data collection (Miles *et al.*, 1988).

Because the majority of studies incorporating stroke volume measurement have been limited to the use of the previously mentioned techniques, stroke volume dynamics during light exercise are unclear. Also the potential risk involved with

stress testing older subjects and the previously invasive nature of the technology for assessing cardiac output have combined to limit research into cardiac response of older subjects during exercise.

In contrast to the previous methods of estimating stroke volume, impedance cardiography allows the assessment of stroke volume safely, simply, non-invasively and on a specified beat-by-beat basis, and therefore, provides an alternative technique in the investigation stroke volume response dynamics during exercise.

Impedance cardiography involves the measurement of thoracic electrical impedance variations that provides a method of estimating stroke volume non-invasively. Impedance cardiography uses methods pioneered by Nyboer (1959), who developed the thoracic tetrapolar electrode arrangement (see Figure 3.1). This type of electrode configuration requires the outer two electrodes to introduce a high frequency current and the inner two voltage electrodes to measure the surface potential which is proportional to impedance and thoracic blood volume changes. Nyboer also proposed the term impedance plethysmography, which refers to the measurement of volume changes in the body, derived from changes in electrical impedance. Because blood is a conductor, each increase in thoracic blood volume following ventricular contraction produces an interpretable change in thoracic resistance. Thus, impedance cardiograph systems induce a constant magnitude, alternating current field along the thorax, measure impedance changes with each heartbeat, and provide an output voltage that can be interpreted as reflecting stroke volume. Kubicek developed and proposed the stroke volume equation (see Methods 3.4.4.3.1 Stroke volume) in the early 1960's (Kubicek *et al.*, 1966) and since 1960, numerous studies have been performed refining and validating these methods (Ebert

et al., 1984; Hatcher, 1986; Sheps *et al.*, 1982; Tanaka *et al.*, 1986; Teo *et al.*, 1985). For example, Ebert *et al.* (1984) reported a high correlation ($r=0.89$, $p<0.001$) between changes in stroke volume measured by simultaneous left ventriculogram and impedance cardiography. Recently, Pickett and Buell (1992) studied 201 pairs of subjects by simultaneously performed thermodilution and impedance cardiography. As a result data indicated that impedance cardiac output values correlated highly ($r=0.75$; $p<0.0001$) with thermodilution values. Impedance cardiography has also been validated against carbon dioxide rebreathing in the current laboratory (see Section 3.4.4.1 for validation of impedance cardiogram). Thus, impedance cardiography is a simple, reliable, non-invasive, indirect method of assessing stroke volume on a beat-by-beat basis throughout submaximal exercise.

There is a paucity of research describing cardiac output measurement of older subjects during exercise using impedance cardiography. However, Williams and Caird (1980) studied 40 subjects with ages ranging from 64-95 years during semi-supine rest. Cardiac output was determined by both impedance cardiography and an isotopic indicator-dilution method. As a result there was no significant difference between the cardiac output results of the two methods. Subsequently this study was extended by Williams and Caird (1985) to include 93 patients. Cardiac output was determined by radionuclide angiography and impedance cardiography. The authors reported excellent agreement between the two methods in patients with sinus rhythm and no regurgitant valvular lesions. The results confirmed the previous study with high correlations ($r=0.90$, $p<0.01$) between the cardiac output values as determined by the two methods. Thus, impedance cardiography has previously been used to

assess stroke volume with older subjects during rest, however, no previous research using impedance cardiography to assess stroke volume with older subjects during exercise appears to have been previously documented.

2.9 SUMMARY

Although the age-associated decline in cardiovascular function is inevitable, the rate of decline may be reduced through endurance training. Endurance trained older males have significantly greater aerobic power compared to that of untrained older males and sedentary young males. Research indicates that the enhanced aerobic power of endurance trained older males is significantly influenced by their ability to increase stroke volume during exercise which consequently permits them to increase cardiac output. However, stroke volume response dynamics of trained and untrained older males during exercise are unclear, mainly due to previous measurement limitations and the paucity of previous research with older subjects during exercise. In the current study the methodology used to investigate these questions was impedance cardiography, a simple non-invasive, safe, beat-by-beat indirect method of estimating stroke volume. Thus, due to the availability of impedance cardiography questions concerning stroke volume response dynamics during varying exercise intensities were able to be investigated. Whether or not stroke volume peaks at lower levels of $\dot{V}O_{2peak}$ than previously reported, whether untrained older males will increase stroke volume during exercise, and the stroke volume response dynamics of trained compared to untrained older males during exercise are the major questions which this study will investigate. An additional aim of the study was to compare the stroke volume response dynamics of endurance trained Runners, to endurance trained Cyclists, during a non-specific modality of

exercise such as cycle ergometry.

3.1 SUBJECTS

Subjects were trained older male Cyclists ($n=10$), trained older Runners ($n=11$), and healthy but untrained older Controls ($n=10$). Inclusion in the study required subjects to be a minimum 60 years of age. Subjects were screened by a cardiac nurse who excluded those with a current pathology (e.g., cardiovascular disease), abnormal cardiac function at rest, and intake of medication that may have influenced exercise performance. Criteria for selection in the trained groups was a current training regime of at least four sessions per week, a past continuous training history of at least five years, and, a $\dot{V}O_{2peak}$ greater than $45 \text{ ml.kg}^{-1}\text{min}^{-1}$. The minimum $\dot{V}O_{2peak}$ criteria for inclusion in the study was based on a previous study by Heath *et al.* (1981) of endurance trained master athletes (average age 59 years) which revealed an average aerobic power of $58.7 \text{ ml.kg}^{-1} \text{ min}^{-1}$. Untrained older males were active members of the community (e.g., golfers, walkers) who were not involved in a regular training program. Subject characteristics are summarized in Table 1. The study was approved by the University of Wollongong Human Experimentation Ethics Committee (see Appendix B) and all subjects provided informed consent.

3.2 TEST ENVIRONMENT

During exercise testing a physician and a cardiac nurse were in attendance. Also a medical trolley equipped with first line emergency drugs (e.g., adrenaline, atropine, and lignocaine), resuscitation equipment, and defibrillator were available.

3.3 EXPERIMENTAL PROTOCOL

3.3.1 Orientation/baseline session

Subjects were required to attend an orientation/baseline session that involved screening (Physical Activity Readiness-Questionnaire [PAR-Q]), 12-lead ECG, and familiarisation with the laboratory and experimental protocol. Also, subjects were requested to read and complete a human subjects consent form and a personal health and exercise history questionnaire. The latter detailing the frequency, intensity, duration, and category of exercise participation, together with any past or current health problems. Finally, subjects were briefly informed of the requirements of the study.

Skinfold thickness were measured at eight sites together with height and mass measurements. While at rest, 12-lead ECG was collected and assessed later by the cardiac nurse for any abnormalities. Two subjects were eliminated from the study due to high blood pressure and suspected left bundle branch block which were detected during screening. Resting blood pressure and heart rate were also recorded. In preparation for the second session, subjects were requested to abstain from eating, smoking, and ingesting caffeine and alcohol for at least 5 hours prior to testing.

3.3.2 Testing session

During the second session subjects were prepared with tetrapolar configuration of impedance electrodes. Voltage electrodes were placed around the base of the neck and around the thorax at the level of the xiphisternal junction. Current electrode bands were placed around the upper part of the neck and around the lower region of the rib cage with a minimum of at least 3 cm separation from the adjacent voltage electrode. Also, three ECG spot electrodes, and the blood pressure cuff were fitted. The impedance cardiograph was then calibrated and ECG

tracings checked. Subjects were required to rest supine for 15 minutes and data was collected during the last 5 minutes and averaged at 30-second intervals. Subjects then sat upright for 5 minutes and data was collected during the last 3 minutes of this period and also averaged at 30-second intervals. The subject was then instructed to mount the ergometer where adjustments to seat and handlebars were made and the headset, mouthpiece, and noseclamp were fitted. The Quinton system was calibrated prior to testing on each occasion.

3.3.3 Exercise protocol

Subjects were instructed to remain in an upright position with the arm used to collect blood pressure by their side and to commence cycling at a cadence of 70 revolutions per minute until volitional exhaustion terminated the test. The initial load was 20 Watts for the first 2 min and was then increased by 1 Watt every 3 seconds thereafter.

In addition, rating of perceived exertion (Borg, 1982) was recorded every minute during the test. At rest and during exercise continuous measurements of cardiac output, stroke volume, and total peripheral resistance were averaged every 30 seconds, whereas blood pressure was recorded automatically each minute. Oxygen consumption and carbon dioxide elimination were collected breath-by-breath during exercise and $\dot{V}O_{2\max}$ was determined as the highest one-minute average oxygen consumption in $L \cdot \min^{-1}$. All subjects received verbal encouragement. During recovery workload was reduced to 50 Watts and heart rate was monitored by the cardiac nurse.

3.4 APPARATUS

3.4.1 Electrocardiogram

During the initial session ECG was recorded with a 12-lead Quinton electrocardiogram (Model number Q750). The exercise electrocardiogram was recorded through a Cardio Life physiograph (Nihon Kohden). The electrocardiogram was recorded using 3-spot electrodes in a Lead II configuration.

3.4.2 Blood pressure

Systolic and diastolic blood pressure were collected and displayed every minute using an automated blood pressure monitor (Quinton, Model 412) and then recorded manually. The cuff was wrapped around the upper right arm with the microphone placed directly over the brachial artery.

3.4.3 Peak oxygen consumption

Peak oxygen consumption was assessed using the Quinton gas analysis system (Model Q-Plex I) that comprised of a Hans Rudolph pneumotachograph (Serial No. 187010), a zirconia oxide oxygen analyzer, and an infra-red carbon dioxide analyzer. Subjects breathed through a Hans Rudolph two-way valve, connected to the Quinton system via low resistance tubing. The valve was held in place by a headset and a nose clamp was fitted.

The Quinton Q-Plex I computerised gas analysis system used in this investigation has previously been validated within the present laboratory (Solomon, 1991) for oxygen and carbon dioxide concentrations, and minute ventilation by comparing expired gas volumes and gas concentrations from the current system to those obtained independently. The partial pressure of oxygen was validated against an Applied Electrochemistry oxygen analyser (model number S-3A) and the carbon dioxide concentrations were validated against a Beckman carbon dioxide analyser (model number LB-2). Minute ventilation was validated using a wedge spirometer

with volumes from both systems adjusted to BTPS conditions. Oxygen uptake and carbon dioxide production determined by the Quinton Q-Plex 1 system has been independently validated against other gas analysis systems (Chypchar *et al.* 1990).

3.4.4 Impedance cardiogram

Cardiac performance was measured non-invasively using impedance cardiography. The impedance cardiogram was recorded by a Minnesota Impedance Cardiograph (Model 304B). The impedance cardiogram was collected using a tetrapolar configuration of electrodes (see Figure 3.1) originally proposed by Nyboer (Miles *et al.*, 1988). A computer-based system processed and recorded the ECG, basal thoracic impedance between the recording electrodes (Z_0 : Figure 3.2), and the first derivative of the pulsatile impedance (the maximum rate of change in the impedance waveform on a given beat (dZ/dt_{\max} : Figure 3.2)). Specialized software using ensemble averaging was used to process the impedance cardiogram (COP, Microtronics Inc., Chapel Hill, NC).

3.4.4.1 Validation of impedance cardiography

Impedance cardiography has been shown to be a valid, reliable method for assessing stroke volume during exercise with young subjects in other laboratories (Hatcher, 1986; Sheps *et al.*, 1982; Tanaka *et al.*, 1986; Teo *et al.*, 1985) and in the present lab (Figure 3.4). Also, during rest with older subjects (Ebert *et al.*, 1984; Williams & Caird, 1985) and exercise with older subjects (Figure 3.5; McLaren *et al.*, 1995). In the present laboratory impedance cardiography was compared during rest and exercise to carbon dioxide rebreathing using a SensorMedics metabolic cart (model 2900). This indirect method to obtain cardiac output uses the Fick equation: $\text{cardiac output} = \dot{V}_{\text{co}_2} / (\text{DCO}_2 \cdot \bar{v} - a)$, in which \dot{V}_{co_2} is

carbon dioxide release in ml/min, $\text{DCO}_2 \cdot \bar{v} - a$ is the difference in mixed-venous to arterial carbon dioxide content. This procedure uses Fick's formula, in which oxygen parameters are substituted by carbon-dioxide parameters to estimate the carbon dioxide content of mixed venous blood during exercise. During this test subjects performed submaximal cycle ergometry at a constant pedalling speed (60 revolutions per minute) and at an intensity progressively increased by .8 kilopond every 4 minutes. This step intensity duration allowed subjects to exercise at a steady-metabolic rate as required by the rebreathing technique (Defares, 1958). While the subject rebreathed, impedance-derived stroke volume values were calculated and compared with the stroke volume obtained by rebreathing. Figures 3.3 and 3.4 show that both techniques gave similar heart rate matched values for cardiac output both at rest and during exercise up to heart rates of $150 \text{ b} \cdot \text{min}^{-1}$ with both young and old subjects.

3.4.4.2 Reliability of impedance cardiogram

The reliability of impedance cardiography was ascertained by assessing cardiac output and stroke volume of 6 older subjects during resting conditions on two separate occasions. Correlation between the two sessions was significant ($r = 0.98$; $p < 0.01$).

3.4.4.3 Impedance cardiogram measures

3.4.4.3.1 Stroke volume

Stroke volume was determined by the Kubicek equation :

$$\text{SV} = \rho \cdot (L/Z_0) \cdot \text{LVET} \cdot dZ/dT \text{ max}$$

where; SV = stroke volume,

ρ = resistivity of blood ($135 \text{ ohm} \cdot \text{sec}^{-1}$),

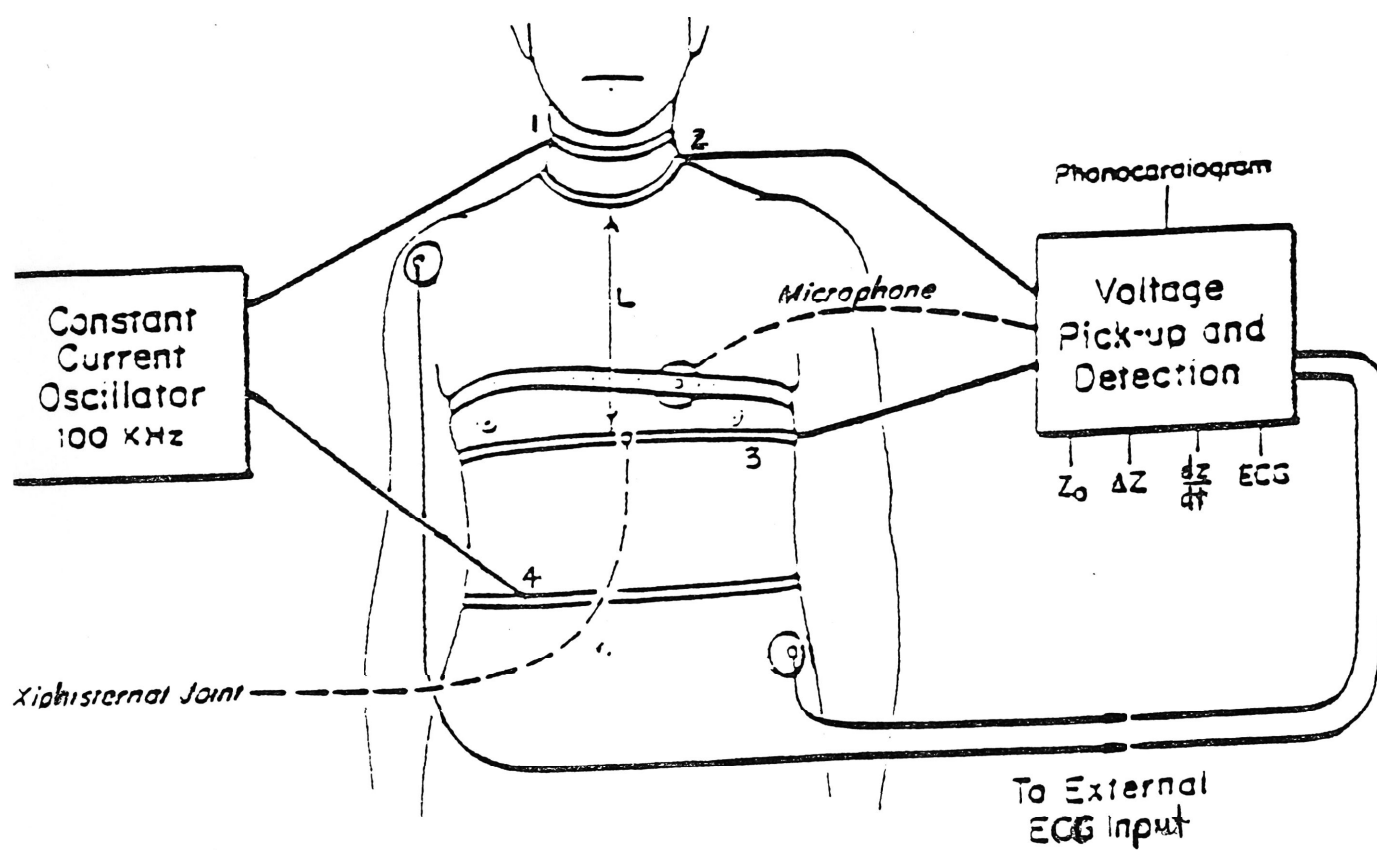


Figure 3.1 Tetrapolar configuration of aluminium electrodes used in impedance cardiography.

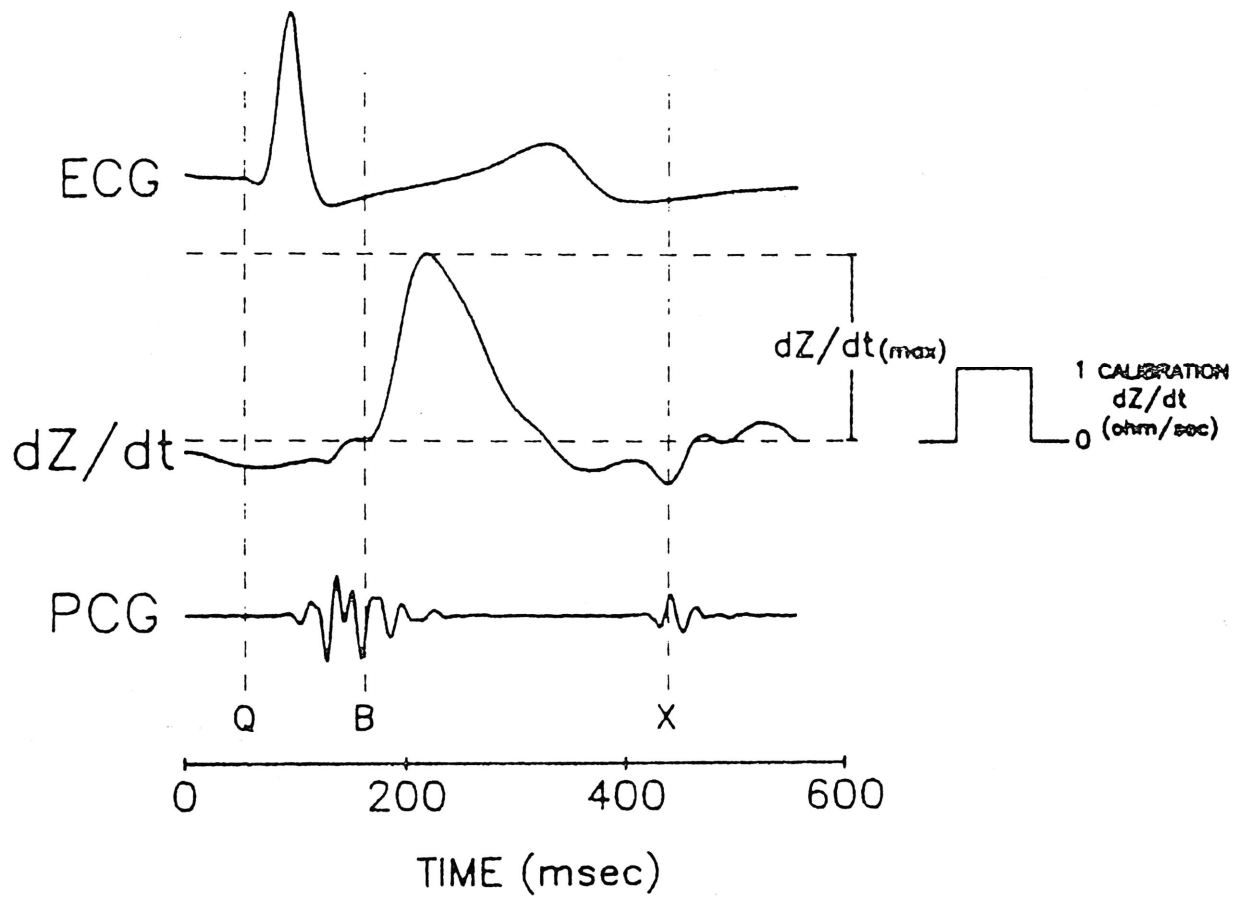


Figure 3.2 Impedance cardiogram waveform components shown are the ECG Q-wave (Q), dz/dt B-point (B), and dz/dt X-point (X). Electrocardiogram (ECG), first derivative of the pulsatile thoracic impedance signal (dz/dt), and phonocardiogram (PCG) recorded during electromechanical systole of a cardiac cycle (from Sherwood *et al.* 1990).

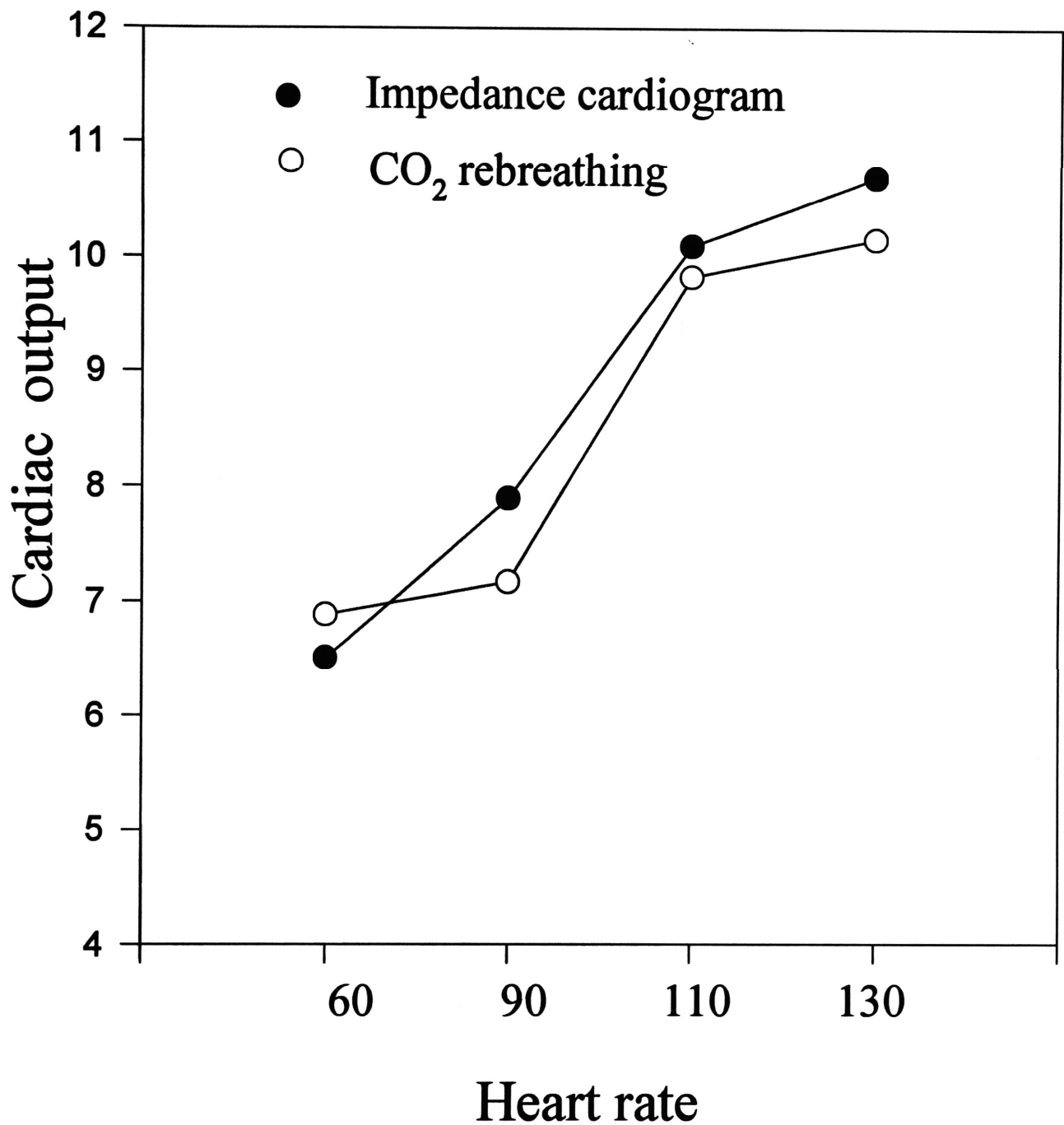


Figure 3.3 Cardiac output-heart rate relationship for a young subject. Cardiac output (litres) determined by carbon dioxide rebreathing and impedance cardiography.

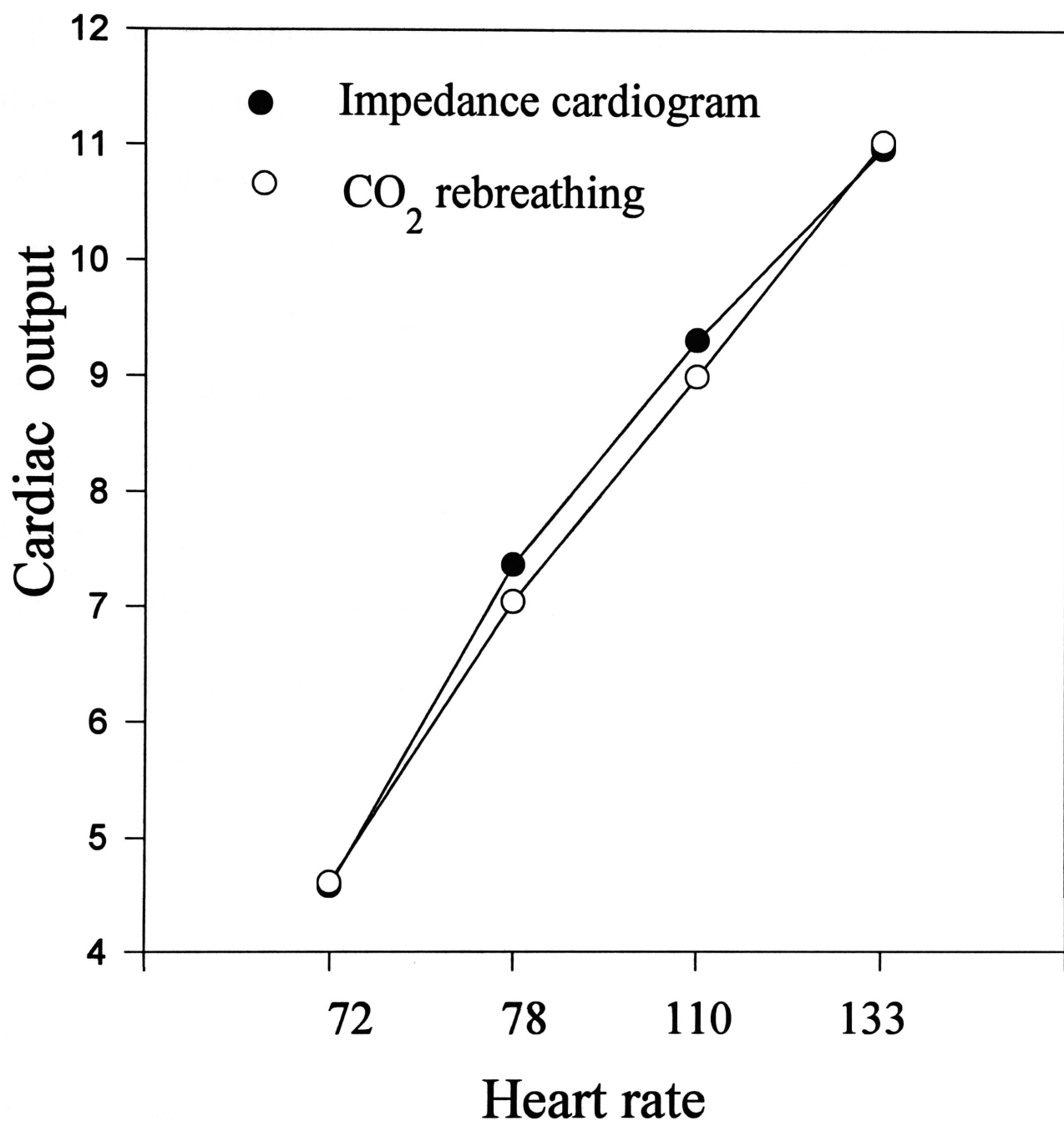


Figure 3.4 Cardiac output-heart rate relationship for an older subject. Cardiac output (litres) determined by carbon dioxide rebreathing and impedance cardiography.

L = distance between voltage electrodes (cm),

Z_0 = basal impedance,

LVET = left ventricular ejection time,

dZ/dT max = maximum rate of change of impedance during systole ($\text{ohm} \cdot \text{sec}^{-1}$).

(Kubicek *et al.*, 1966).

3.4.4.3.2 Cardiac Output

Cardiac output was computed by the COP software based on the equation:

Cardiac output = stroke volume x heart rate.

3.4.4.3.3 Pre-ejection period

Pre-ejection period was computed as the interval from the ECG Q wave onset to dZ/dT B point in milliseconds (Sherwood *et al.*, 1990; Figure 3.2).

3.4.4.3.4 Left-ventricular ejection time

Left ventricular ejection time was computed as the interval from dZ/dT B point to dZ/dT X point in milliseconds (Sherwood *et al.*, 1990; Figure 3.2).

3.4.5 Electronic-braked cycle ergometer

During the exercise test external work was assessed by a Lode electronically-braked cycle ergometer (Excalibur Sport).

3.5 MEASURES

3.5.1 Anthropometric

Body fat was assessed from skinfold measures at eight sites (abdominal, triceps, suprailiac, midaxillary, thigh, calf, biceps, and subscapula). Body density was calculated using the formula of Jackson and Pollock (1978) and percent fat from the formula of Siri (1961).

3.5.2 Heart rate

Heart rate was determined from the ECG recording as the mean interbeat-interval between the peak voltage of R waves.

3.5.3 Peak oxygen consumption

$\dot{V}O_{2peak}$ was determined as the highest one-minute average oxygen consumption in $L \cdot min^{-1}$. The end point was achieved when the subject was unable to continue because of volitional fatigue. Other indicators included heart rate at age-estimated maximum, plateau of oxygen consumption, and a respiratory exchange ratio greater than 1.10.

3.5.4 Systolic/diastolic blood pressure

Both systolic and diastolic blood pressure were recorded automatically every minute using the Quinton monitor. Automatic continuous blood pressure monitoring provides an efficient alternative to traditional auscultation cuff methods and assists in reducing experimenter error.

3.5.5 Mean arterial pressure

Mean arterial pressure was calculated using the COP software based on the equation: $1/3 \times \text{pulse pressure (systolic pressure-diastolic)} + \text{diastolic pressure}$.

3.5.6 Total peripheral resistance

The combination of impedance cardiography with the blood pressure monitoring allowed the determination of total peripheral resistance. Total peripheral resistance represents the resistance of the vasculature to blood flow and was computed according to the equation:

$$TPR (\text{dyne} \cdot \text{seconds} \cdot \text{cm}^{-5}) = \text{mean arterial pressure} / \text{cardiac output} \times 80.$$

3.5.7 Rate pressure product

Rate pressure product is linear to myocardial oxygen consumption and was

computed according to the equation:

$$\text{rate pressure product} = \text{systolic blood pressure} \times \text{heart rate}/100.$$

3.6 DATA PROCESSING

3.6.1 Impedance cardiogram

Impedance cardiograms were initially processed using ensemble averaging to filter artifact from the impedance cardiogram. Each data point was edited via the edit mode of the COP software. Data for blood pressure (systolic and diastolic) was entered via the edit mode to enable mean arterial pressure and total peripheral resistance to be calculated.

3.6.2 Statistical analysis

The design of the study included both between and repeated measures. The between factor was group (trained Cyclists versus trained Runners, versus untrained Controls) and the repeated measure was time for each of the measures. Analysis was conducted using the SPSSPC statistical package.

Temporal changes of heart rate, stroke volume, cardiac output, stroke index, cardiac index, total peripheral resistance, rate pressure product, and blood pressure, were compared using between and repeated measures analysis of variance. These variables (apart from $\dot{V}O_{2\text{peak}}$ and blood pressure) were calculated by the COP software (edit mode) at supine and upright resting conditions, and during progressive cycle ergometry. Differences between the three groups for resting values and $\dot{V}O_{2\text{peak}}$ were compared using one-way analysis of variance. A probability of $p < 0.05$ was considered significant. Comparisons were made at heart rates of 90, 100, 110, 120, 130, 140, and 150 $\text{b} \cdot \text{min}^{-1}$. Means and standard error of the mean for all variables at rest and during exercise are reported in Appendix C.

To examine differences in subjects' physical characteristics, one-way analysis of variance with Newman-Keuls post hoc tests were performed. A 3 (Group: Cyclists, Runners, and Controls) X 4 (Time: supine and upright rest) mixed design was used to examine cardiovascular variables at rest. A 3 (Group: Cyclists, Runners, and Controls) X 7 (Time: heart rates during exercise at 90, 100, 110, 120, 130, 140, and 150 b.min⁻¹) mixed design was used to examine cardiovascular differences during exercise.

For analyses that involved repeated measures the conservative *F*-test correction for degrees of freedom (Geisser & Greenhouse, 1958) was applied when symmetry assumptions were violated. When this occurred degrees of freedom for the *F*-statistic were halved and probability values calculated on these revised values. All data distributions were examined and all assumptions for each statistical analysis were tested.

CHAPTER FOUR: RESULTS

Results of this study are divided into three sections. The first section summarises subjects' physical characteristics, whereas the remaining two sections describe aerobic power, and baseline and exercise cardiovascular response.

4.1 SUBJECT CHARACTERISTICS

Subject characteristics are shown in Table 1. The three groups possessed similar age, body surface area, and body mass ($p > 0.05$), whereas inter-group difference existed for height, sum of skinfolds, and resting heart rate. Runners were significantly taller than Cyclists. Also the sum of 8 skinfolds and percent body fat of the Controls was significantly greater than that of Cyclists and Runners. Resting heart rate of the Runners and Cyclists were significantly lower than that of Controls, $F(2, 28) = 6.92, p = .004$.

4.2 AEROBIC POWER AND WORK

Aerobic power for the three groups is illustrated in Table 1. As expected, $\dot{V}O_{2peak}$ expressed in absolute terms, of Cyclists and Runners was significantly higher than that of Controls. Similarly, relative $\dot{V}O_{2peak}$ of Cyclists and Runners was significantly higher than that of Controls. Also, Cyclists had significantly higher relative $\dot{V}O_{2peak}$ than that of Runners. This was reflected in external power production, with Cyclists and Runners generating significantly greater power, $F(12, 168) = 9.77, p = .0001$, at equivalent heart rates right through the exercise protocol. The peak cycle ergometer power output of Cyclists (228 Watts) and Runners (222 Watts) was significantly higher than that of Controls (137 Watts; Figure 4.1).

Table 1: Physical characteristics of the subjects. Data are means with standard errors of the means in parenthesis.

Variable	Cyclists (n=10)	Runners (n=11)	Controls (n=10)
Age (yr)	65.2 (2.1)	65.09 (3.47)	65.50 (1.16)
Height (cm)	171.74 (2.46)	179.05 (6.5) ^a	174.62 (1.77)
Mass (kg)	72.83 (3.13)	74.05 (7.64)	80.71 (2.34)
Body surface area (m ²)	1.85 (.053)	1.91 (.039)	1.96 (.032)
$\dot{V}_{O_{2peak}}$ (L.min ⁻¹)	3.91 (.21) ^b	3.52 (.36) ^b	2.33 (.08)
$\dot{V}_{O_{2peak}}$ (ml.kg ⁻¹ min ⁻¹)	53.75 (1.58) ^{b c}	47.95 (3.94) ^b	28.42 (1.26)
Resting heart rate (b.min ⁻¹)	56.2 (1.26) ^d	50.9 (2.57) ^d	67.65 (3.43)
Sum of 8 skinfolds (mm)	69.03 (5.2)	70.35 (3.64)	92.79 (2.66) ^e
Body fat (%)	12.8 (.82)	12.45 (.51)	15.3 (.59) ^e

Abbreviations: $\dot{V}_{O_{2peak}}$ = peak oxygen consumption;

^a = significantly greater than Cyclists, p<0.05;

^b =significantly greater than Controls p<0.05;

^c = significantly greater than Runners p<0.05;

^d = significantly lower than Controls p<0.05;

^e = significantly greater than Cyclists and Controls p<0.05.

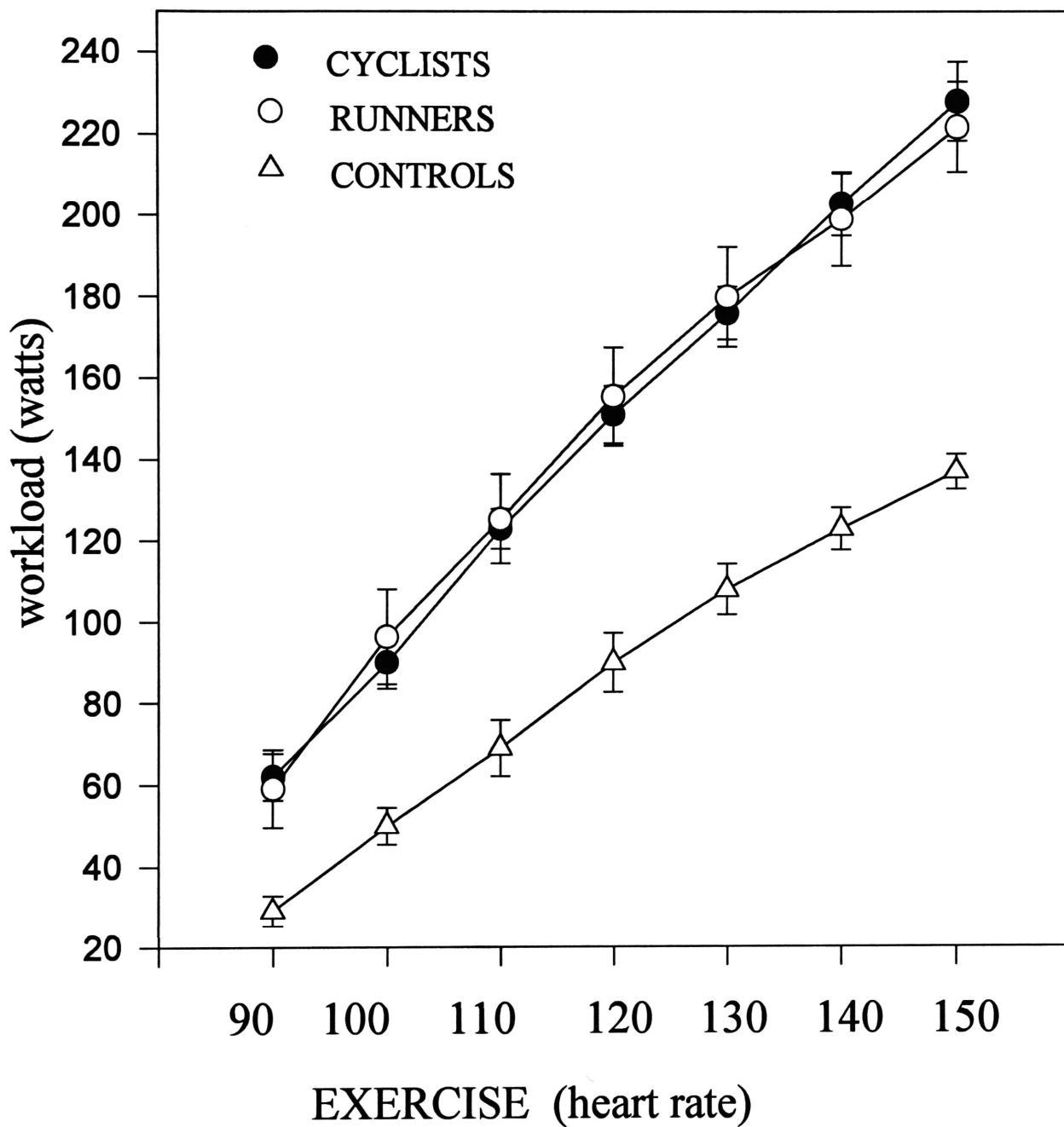


Figure 4.1 Power output in Watts at various heart rates during exercise. Data are means with standard errors of the means.

4.3 BASELINE AND EXERCISE CARDIOVASCULAR RESPONSE

4.3.1 Stroke volume

Analysis on supine and upright resting stroke volume revealed a significant Group main effect, $F(2, 28) = 4.20$, $p = .025$. Post hoc analysis indicated that stroke volume of the Runners at supine rest was significantly higher than that of Controls, while the difference between the two trained groups were not significant ($p > 0.05$; Figure 4.2). From the resting supine stroke volume, all groups experienced a stroke volume reduction when assuming an upright posture. These decrements were similar between groups, approximately 30.7%, 28.3%, and 29%, for Cyclists, Runners, and Controls, respectively. At the commencement of exercise, Cyclists, Runners, and Controls each underwent a stroke volume increase of 41%, 47%, and 31% respectively, at a heart rate of 90 b.min⁻¹. Also, during the initial stages of exercise (heart rates of 90 and 100 b.min⁻¹), stroke volume of Runners (113 ml and 112 ml) was significantly higher compared to that of Cyclists (90 ml and 96 ml). During exercise a significant Group main effect was observed, $F(2, 28) = 6.69$, $p = .004$. Post hoc tests showed that stroke volume of Runners was significantly larger than that of Controls throughout exercise. There was also a significant main effect of Time, $F(3, 84) = 131.62$, $p = .0001$. No significant effect was observed for the Group by Time interaction.

4.3.2 Stroke index

Analysis on supine and upright resting stroke index revealed a significant Group main effect, $F(2, 28) = 4.13$, $p = .027$. Post hoc analysis indicated that stroke index of the Runners (55.5 ml/m²) at supine rest was significantly higher than that of Controls (44.5 ml/m²; Figure 4.3). During exercise a significant Group main

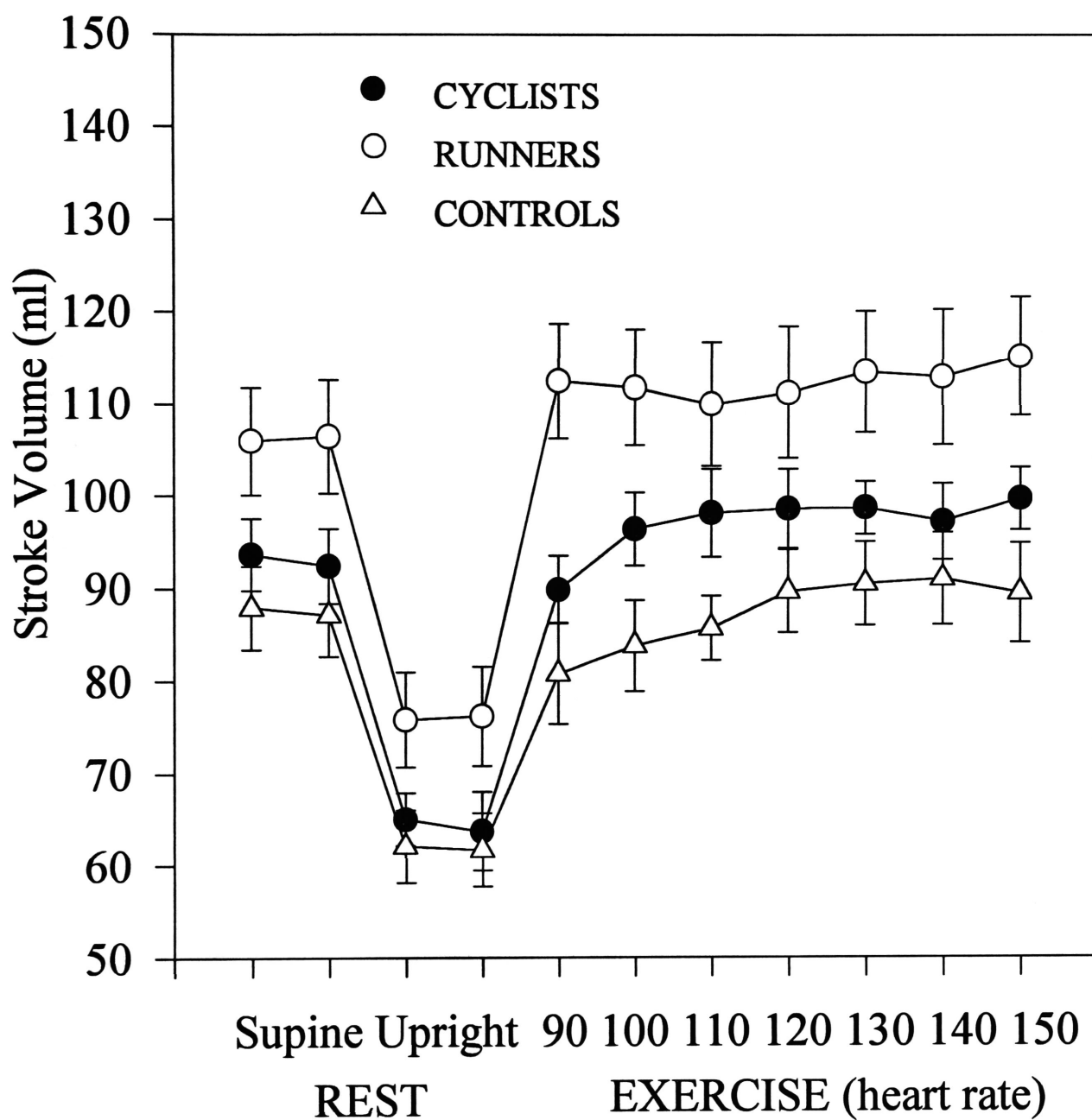


Figure 4.2 Stroke volume at rest and at increasing heart rates during exercise. Data are means with standard errors of the means.

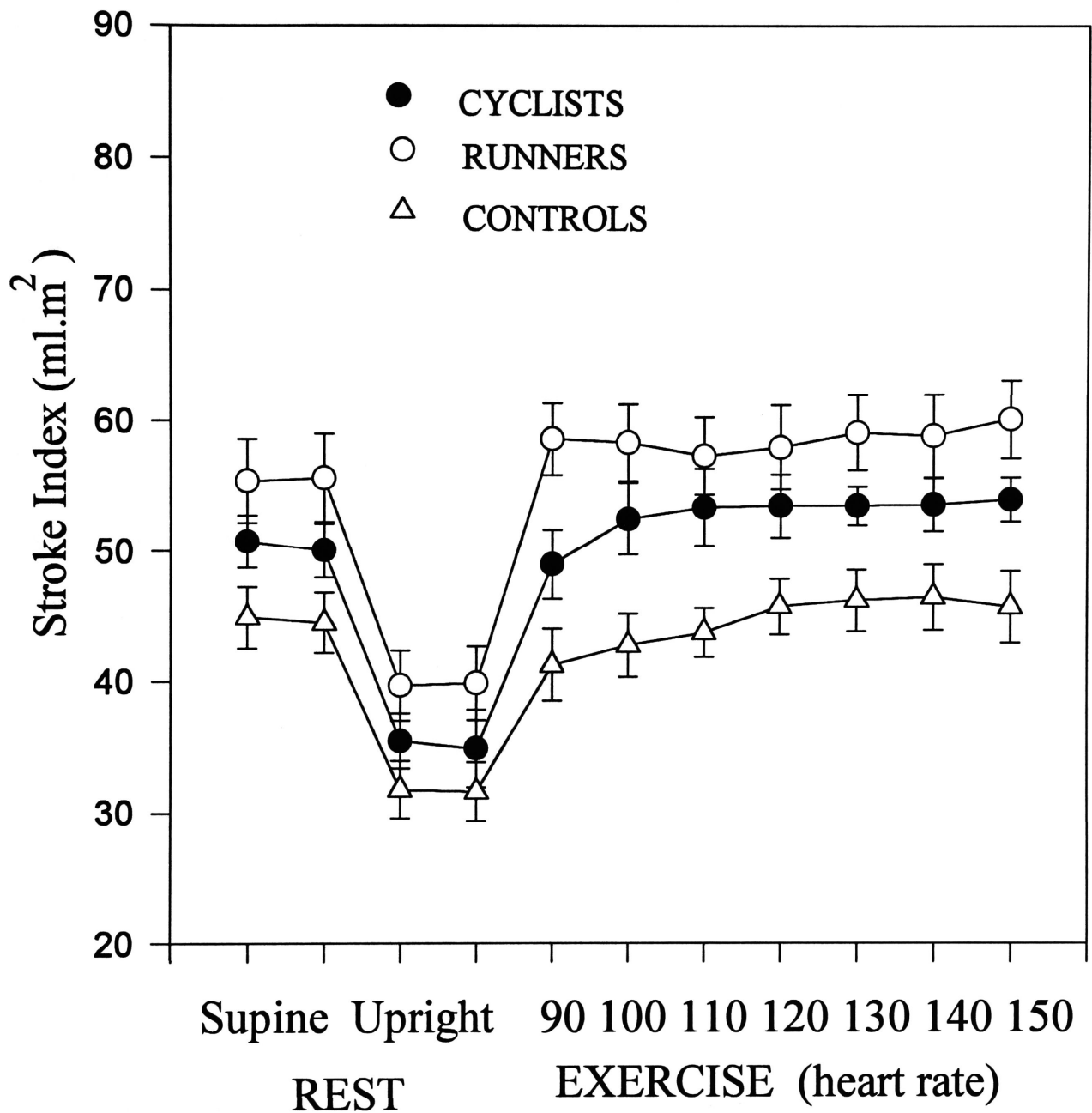


Figure 4.3 Stroke index at rest and various heart rates during exercise. Data are means with standard errors of the means.

effect was also observed, $F(2, 228) = 8.7, p=.001$. Post hoc tests showed that stroke index of Runners was significantly larger than that of Controls throughout exercise. The stroke index of Cyclists was also greater than Controls at 100, 110, 130, and 150 b.min⁻¹. In addition, stroke index of Runners (58.6 ml/m²) was significantly higher than that of Cyclists (49 ml/m²) at a heart rate of 90 b.min⁻¹. There was also a significant main effect of Time, $F(6, 168) = 3.40, p=.0003$. No significant effect was observed for the Group by Time interaction.

4.3.3 Cardiac output

During exercise a significant Group main effect was observed, $F(2, 28) = 6.69, p=.004$. Post hoc tests showed that cardiac output of Runners was significantly larger than that of Controls throughout exercise (Figure 4.4). Also, cardiac output of Runners (10.2 L) was significantly higher than that of Cyclists (8.3 L) at a heart rate of 90 b.min⁻¹. There was also a significant main effect of Time, $F(6, 168) = 148.35, p=.0001$. No significant effect was observed for the Group by Time interaction.

4.3.4 Cardiac index

During exercise a significant Group main effect was also observed, $F(2, 28) = 6.69, p=.004$. Post hoc tests showed that the cardiac index of Runners was significantly larger than that of Controls throughout exercise (Figure 4.5). Also, Runners displayed significantly larger cardiac index (5.3 L/min/m²) than that of Cyclists (4.5 L/min/m²) at a heart rate of 90 b.min⁻¹. There was also a significant main effect of Time, $F(6, 168) = 158.9, p=.0001$. No significant effect was observed for the Group by Time interaction.

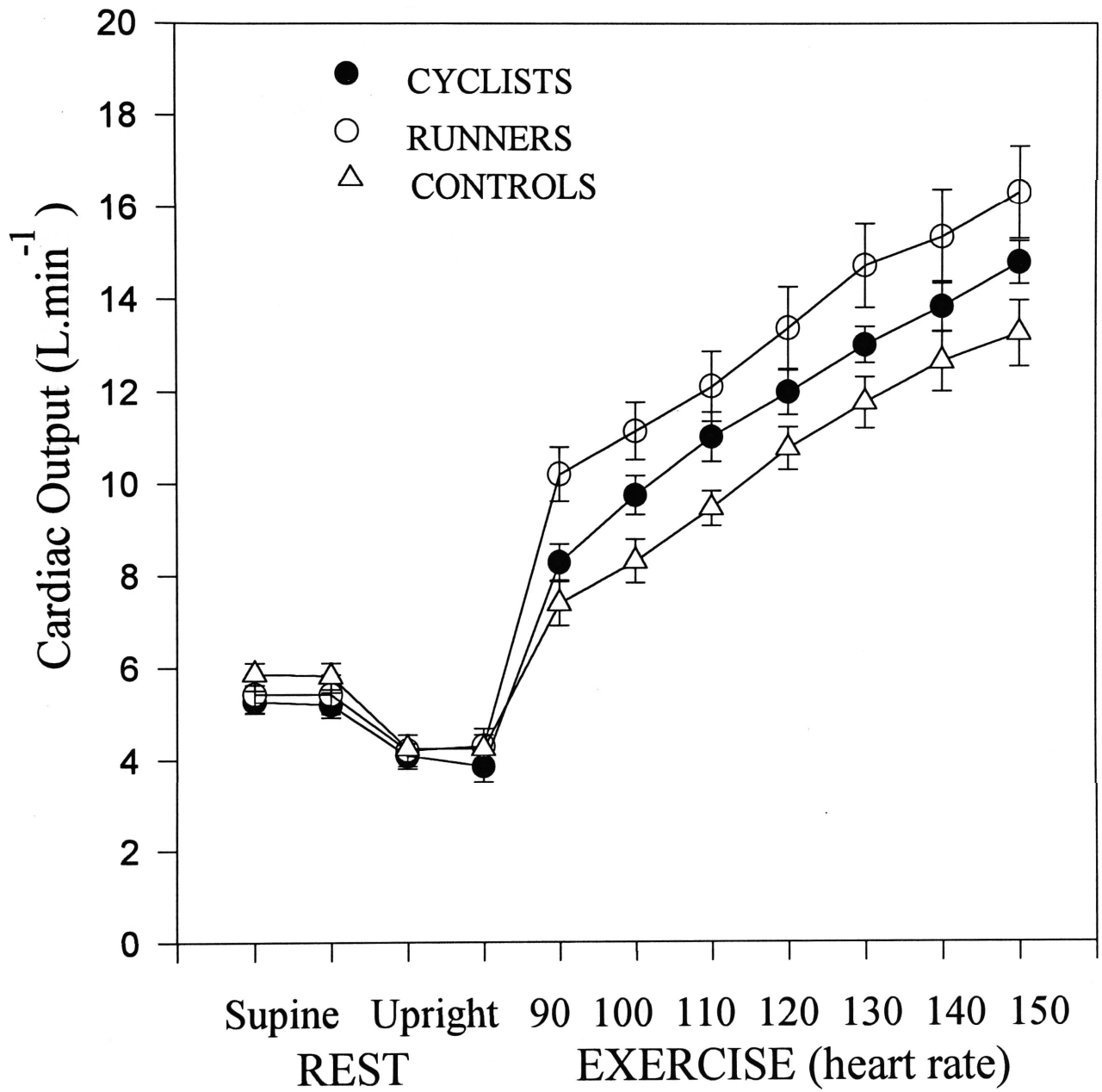


Figure 4.4 Cardiac output at rest and various heart rates during exercise.

Data are means with standard errors of the means.

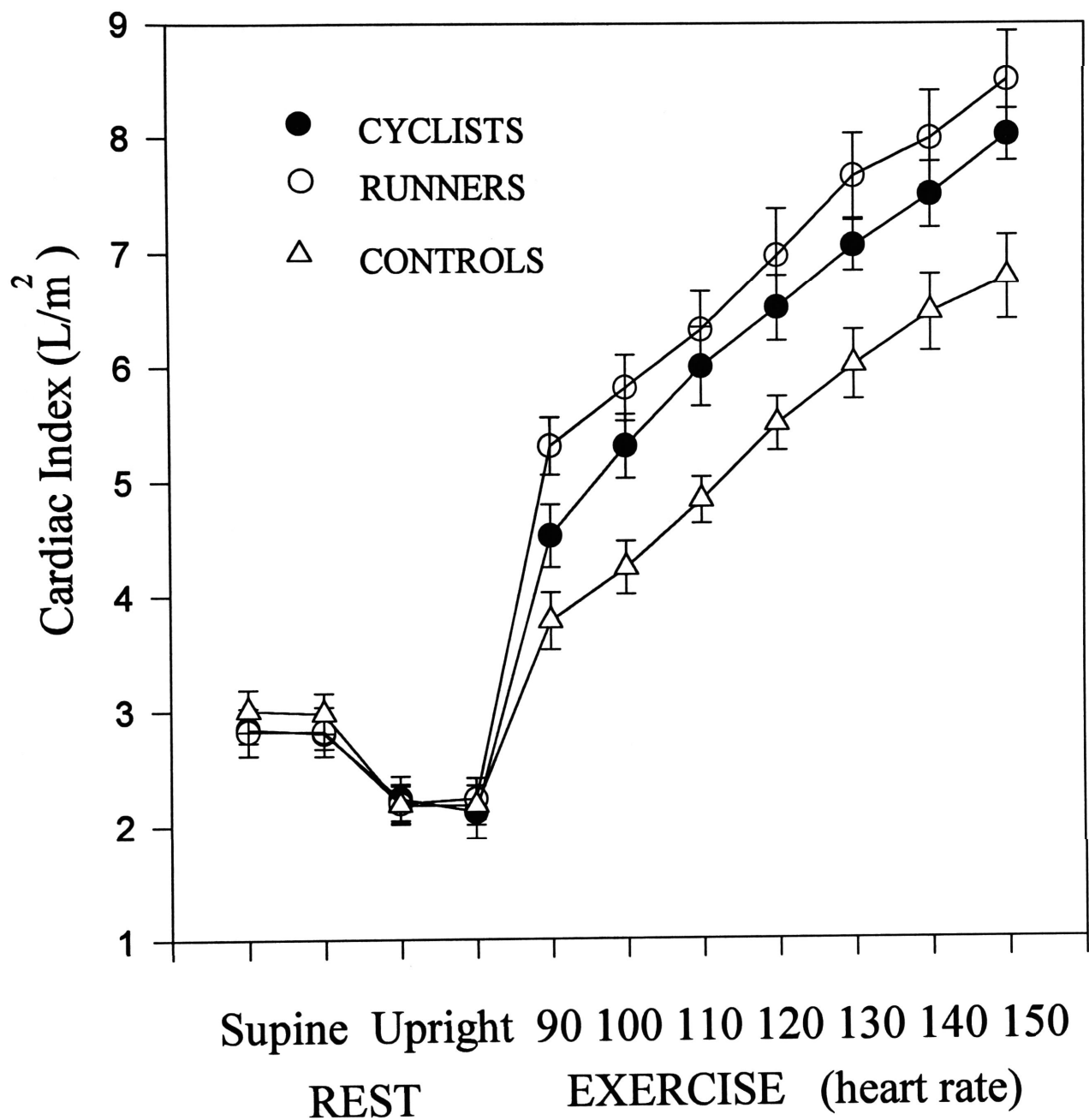


Figure 4.5 Cardiac index at rest and various heart rates during exercise. Data are means with standard error of the means.

4.3.5 Total peripheral resistance

Analysis on supine and upright resting total peripheral resistance revealed no significant Group main effect. During exercise a significant Group main effect was observed between groups, $F(2, 28) = 6.69$, $p = .004$. Post hoc tests revealed that total peripheral resistance of Runners was significantly lower than that of Cyclists and Controls throughout exercise (Figure 4.6). There was also a significant main effect of Time, $F(3, 84) = 131.62$, $p = .0001$. No significant effect was observed for the Group by Time interaction.

4.3.6 Rate pressure product

Analysis on supine and upright resting rate pressure product revealed a significant Group main effect, $F(2, 28) = 5.28$, $p = .011$. Post hoc analysis indicated that rate pressure product of Controls at supine rest was significantly greater than Cyclists and Runners (Figure 4.7). During exercise a significant main effect was also observed between groups, $F(2, 28) = 6.00$, $p = .007$. Post hoc tests showed that Cyclists displayed a greater rate pressure product response compared to that of Runners and Controls during exercise. There was also a significant main effect of Time, $F(6, 168) = 112.94$, $p < .0001$. Cyclists compared to Controls displayed lower rate pressure product at baseline, however, Cyclists possessed significantly greater rate pressure product during exercise (Figure 4.7). No significant effect was observed for the Group by Time interaction.

4.3.7 Systolic blood pressure

Analysis on supine and upright resting systolic blood pressure revealed no significant Group main effect. During exercise a significant main effect was observed between groups, $F(2, 28) = 4.26$, $p = .024$. Post hoc tests showed that

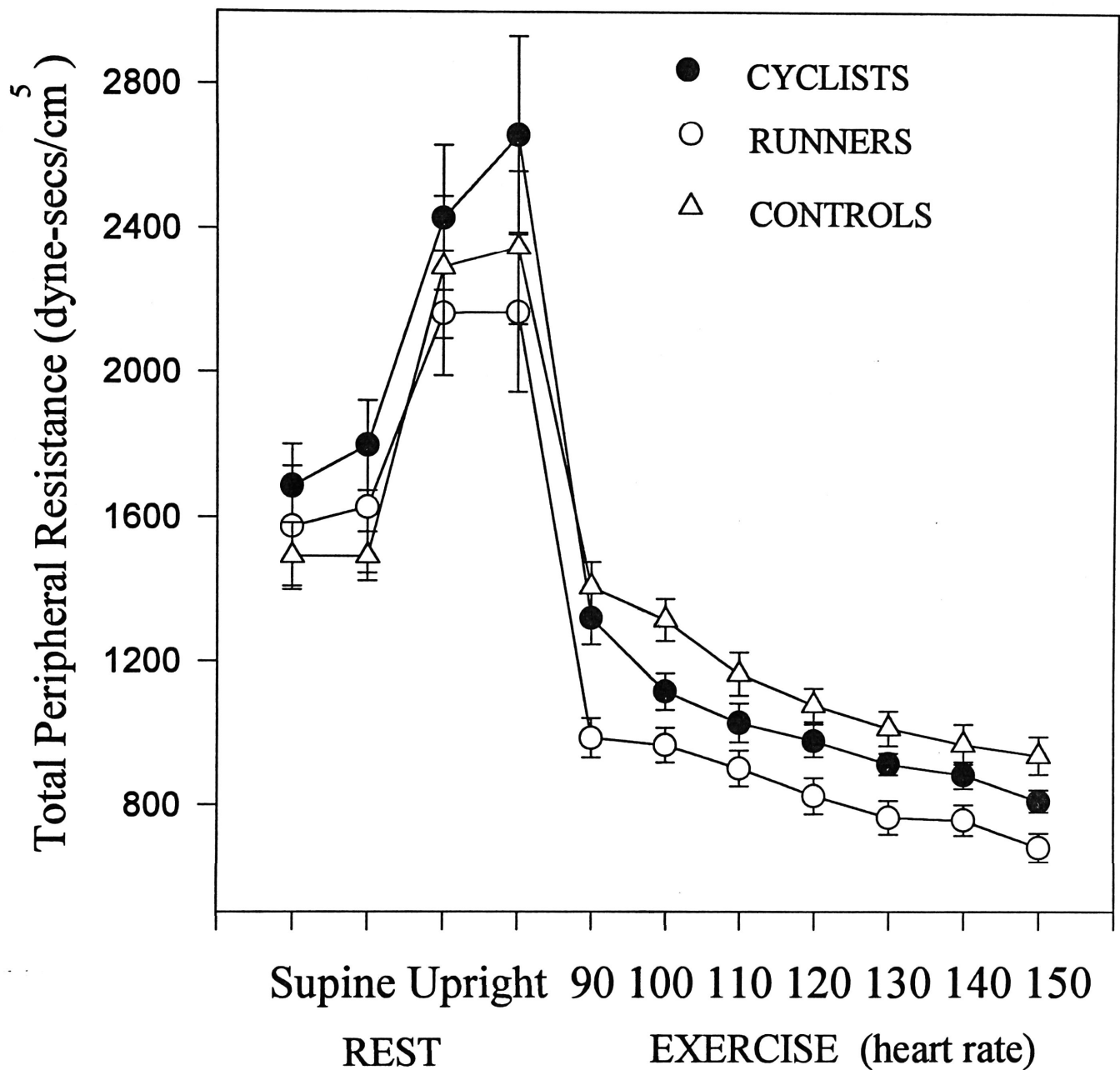


Figure 4.6 Total peripheral resistance at rest and various heart rates during exercise. Data are means with standard errors of the means.

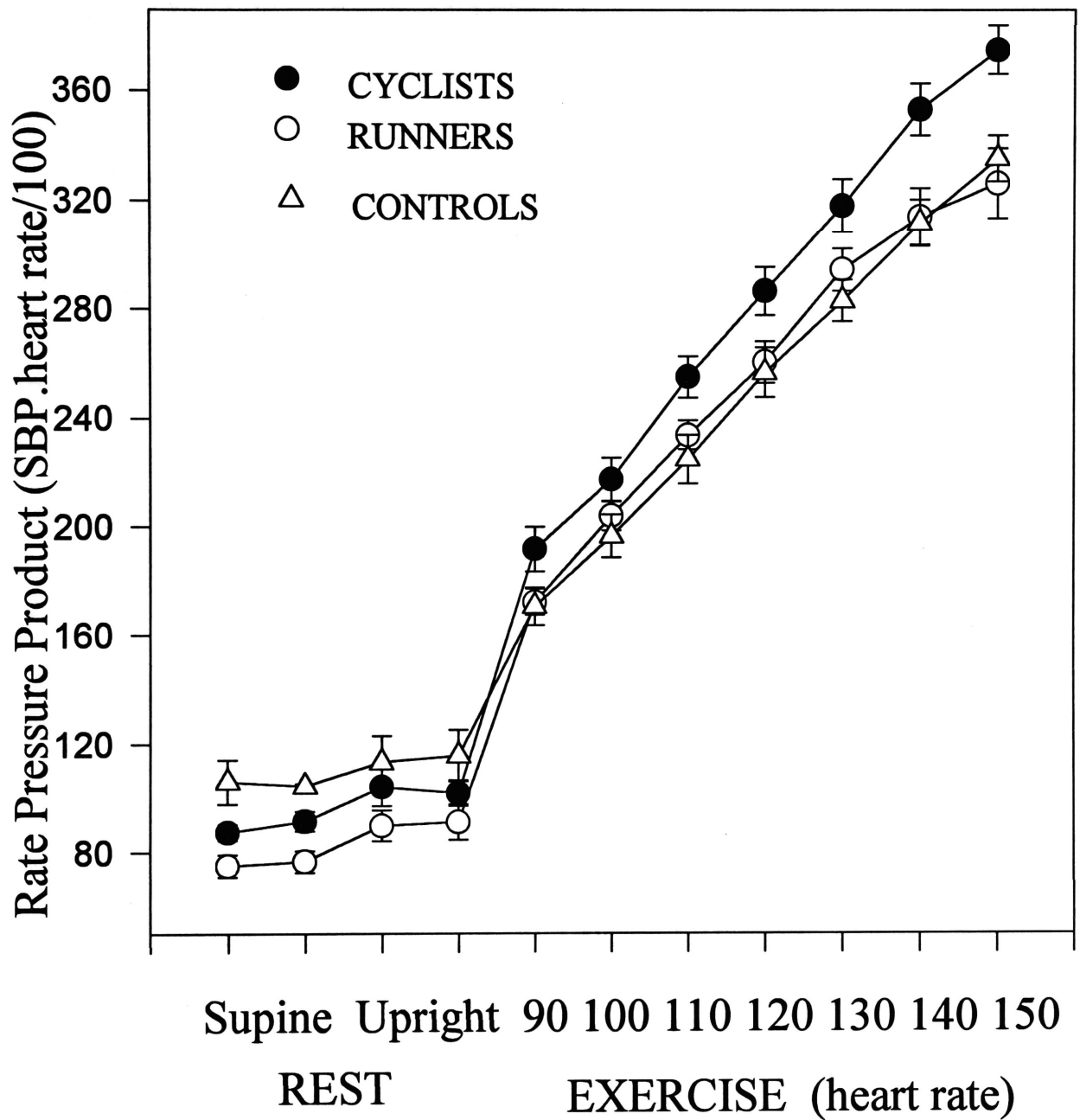


Figure 4.7 Rate pressure product at rest and various heart rates during exercise.

Data are means with standard errors of the means.

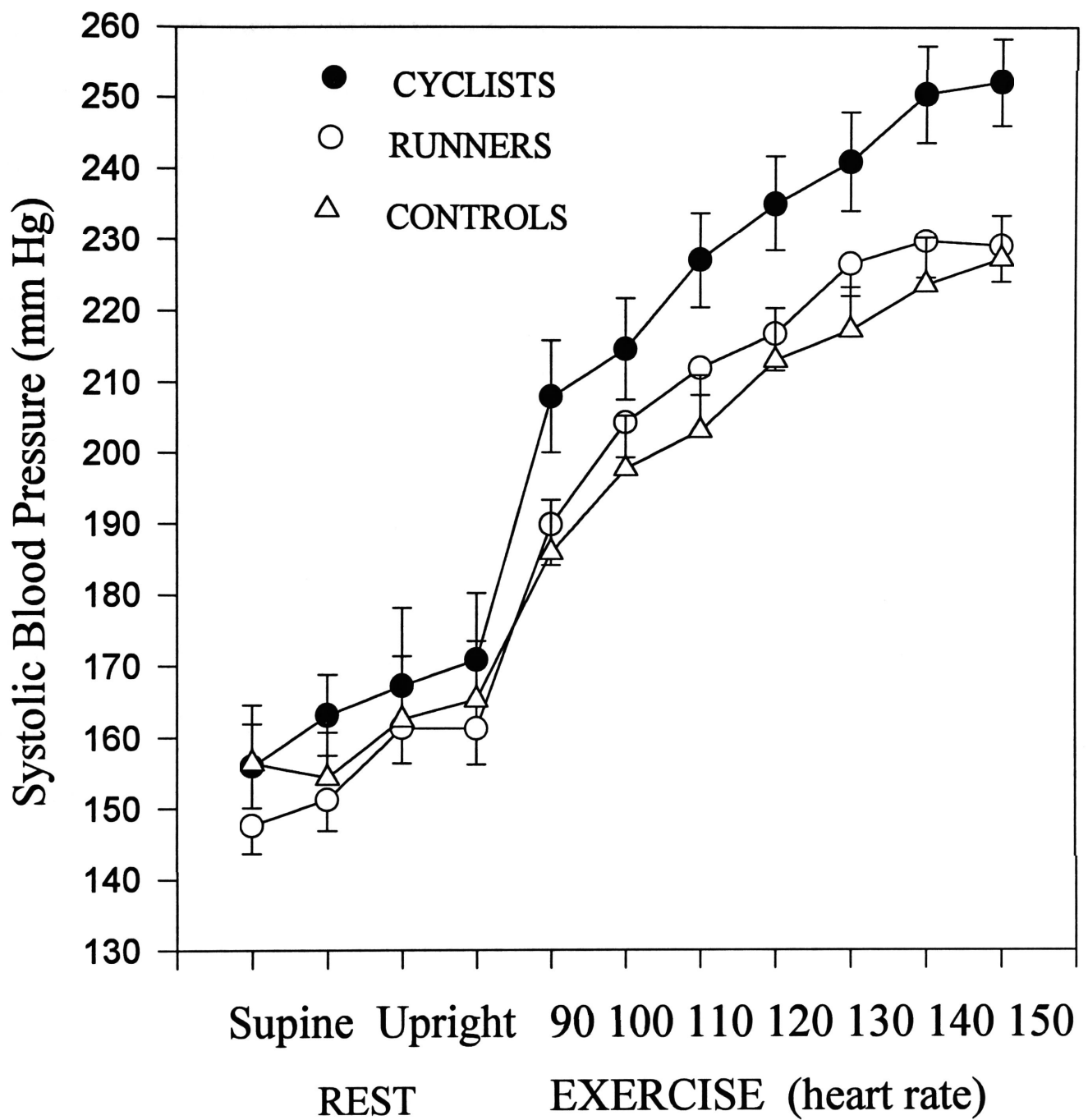


Figure 4.8 Systolic blood pressure at rest and increasing heart rates during exercise. Data are means with standard errors of the means.

Cyclists displayed significantly higher systolic blood pressure than Controls during exercise (Figure 4.8). Cyclists also displayed higher systolic blood pressure than Runners at heart rates of 140 and 150 b.min⁻¹. No significant effect was observed for the Group by Time interaction.

4.3.8 Diastolic blood pressure

Analysis on supine and upright resting diastolic blood pressure revealed no significant Group main effect. During exercise there was no significant difference in diastolic blood pressure between Cyclists, Runners, and Controls (Figure 4.9).

4.3.9 Mean arterial blood pressure

Analysis on supine and upright resting mean arterial blood pressure revealed no significant Group main effect or Group by Time interaction. During exercise there was no significant difference in mean arterial blood pressure between Cyclists, Runners, and Controls (Figure 4.10).

4.3.10 Left-ventricular ejection time

Analysis on supine and upright resting left ventricular ejection time indicated a significant Group main effect, $F(2, 28) = 4.20$, $p = .025$. Post hoc analysis revealed that at supine rest both Cyclist and Runners had significantly longer left ventricular ejection time than Controls (Figure 4.11).

4.3.11 Pre-ejection Period

Analysis on pre-ejection period scores indicated no significant differences during rest or exercise (Figure 4.12).

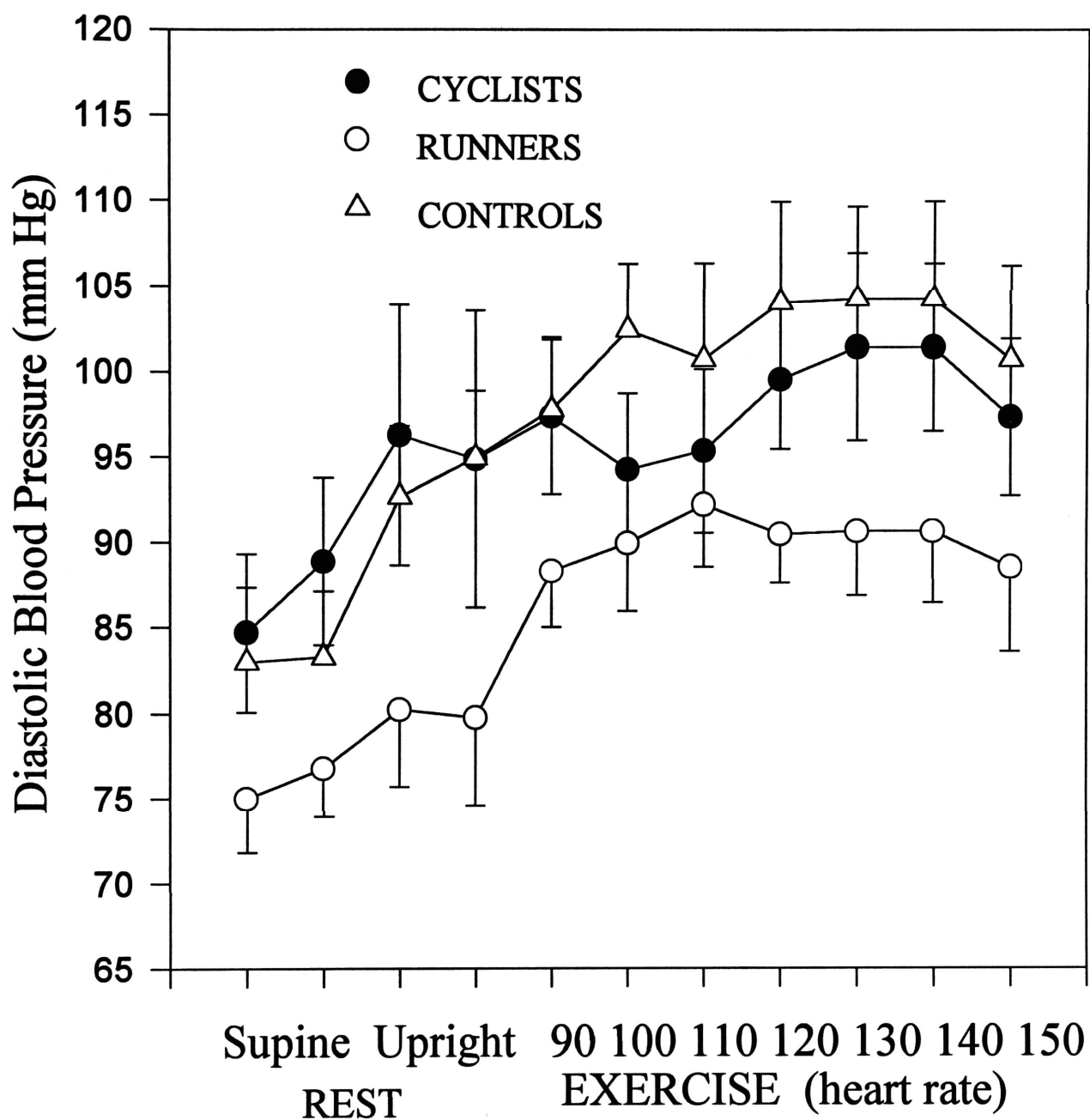


Figure 4.9 Diastolic blood pressure at rest and various heart rates during exercise.

Data are means with standard errors of the means.

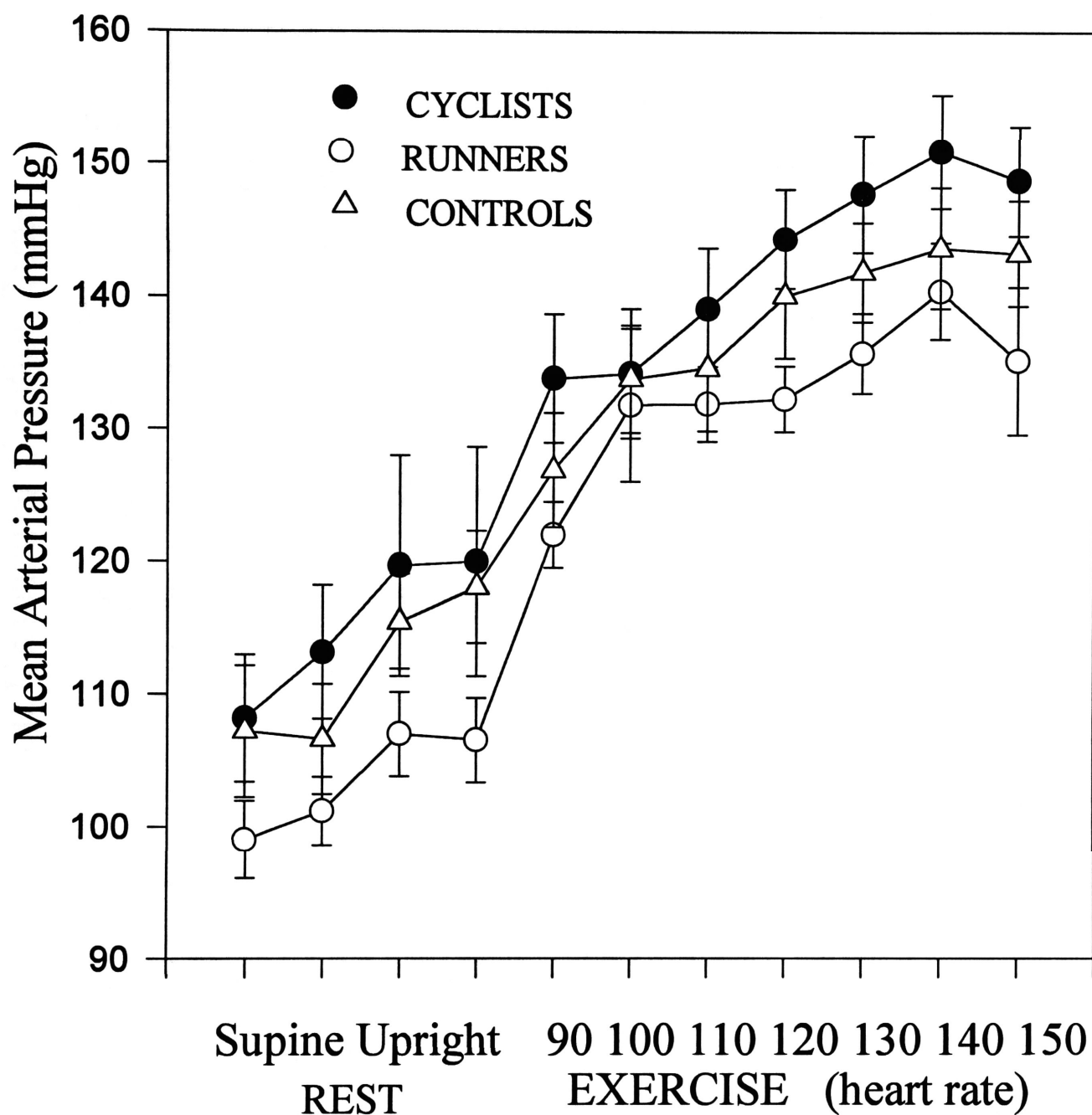


Figure 4.10 Mean arterial blood pressure at rest and various heart rates during exercise. Data are means with standard errors of the means.

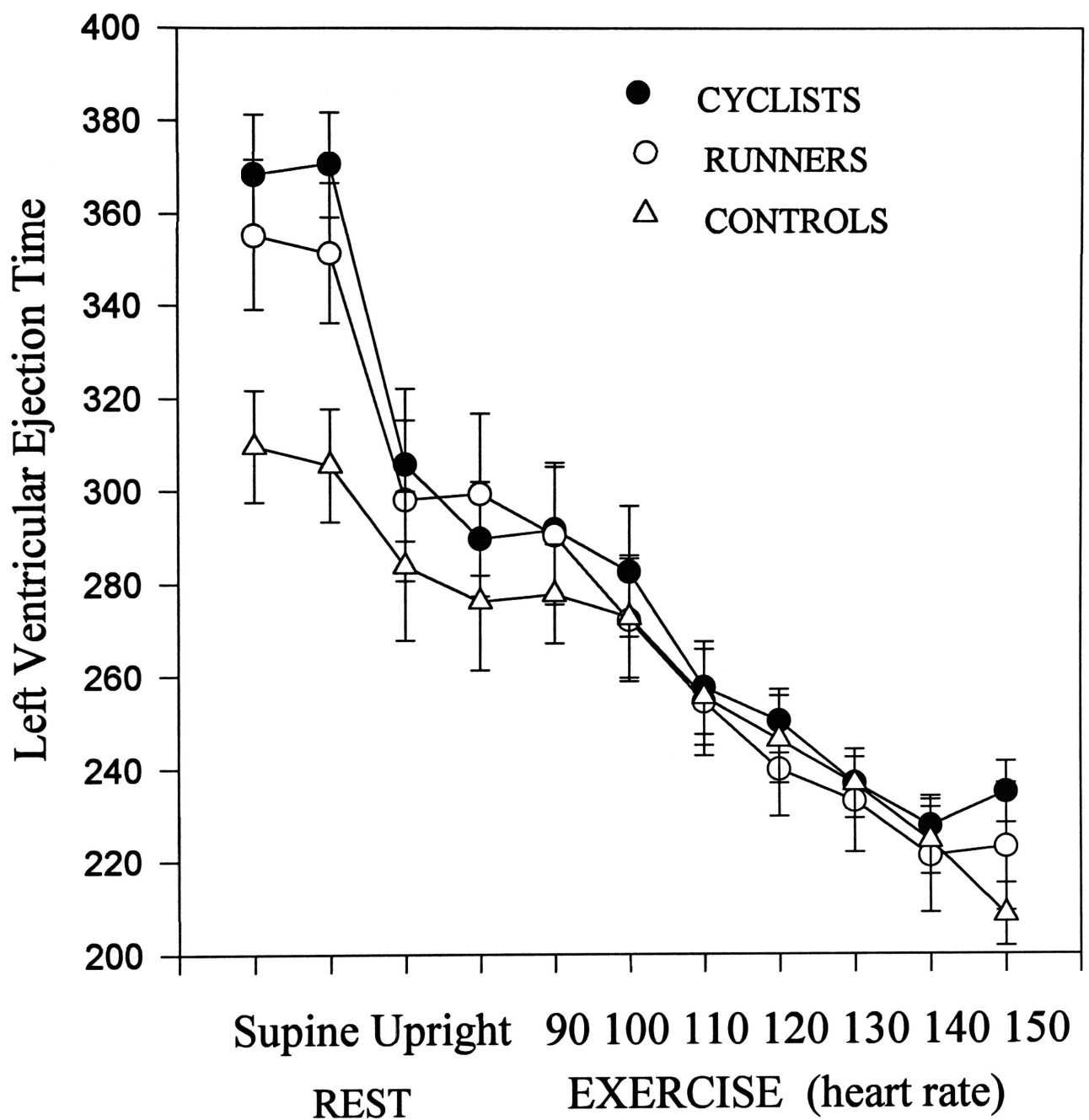


Figure 4.11 Left-ventricular ejection time at rest and various heart rates during exercise. Data are means with standard errors of the means.

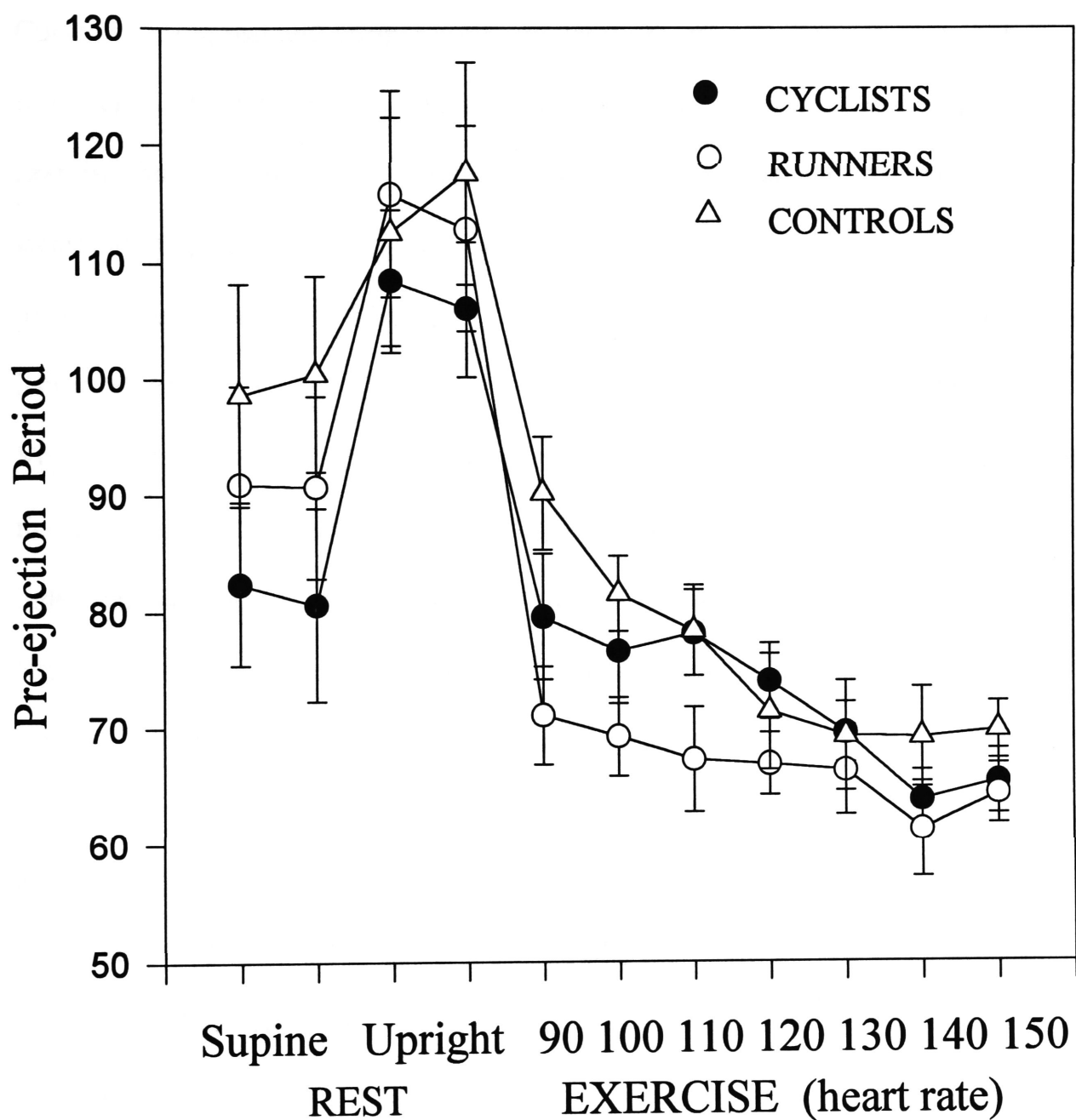


Figure 4.12 Pre-ejection period at rest and various heart rates during exercise.

Data are means with standard errors of the means.

CHAPTER FIVE: DISCUSSION

The main purpose of this study was to compare the stroke volume response of trained older Cyclists, trained older Runners, and healthy untrained Controls during rest and upright cycle ergometry exercise. The major findings were: 1) in all three groups stroke volume was significantly elevated during exercise, however, the stroke volume response of trained older males was significantly greater than untrained Controls; 2) in all three groups stroke volume peaked at less than 20% of $\dot{V}O_{2peak}$; 3) untrained but healthy older males displayed an increased stroke volume during the early stages of exercise; 4) despite a lower $\dot{V}O_{2peak}$ than the Cyclists, Runners displayed a significantly higher stroke volume than that of the Cyclists; 5) at rest Runners displayed larger stroke volume and stroke index compared to Controls; 6) Runners and Cyclists at rest had significantly longer left-ventricular ejection time and lower resting heart rate compared to controls. Finally, at rest the rate pressure product of Controls was higher than that of Cyclists and Runners. This discussion will focus initially upon the stroke volume response of all three groups, the peaking of stroke volume, and the stroke volume dynamics of the untrained Controls. In addition, cardiac adaptations to cross-training, cardiac output and peripheral resistance, and cardiovascular variables at rest will be discussed.

5.1 MAJOR FINDINGS

5.1.1 Stroke volume response

In all three groups stroke volume was significantly elevated during exercise but the stroke volume response of Runners was significantly greater than that of Controls. These findings agree with previous research (Boutcher *et al.*, 1994) that

has shown that young trained cyclists, compared to untrained subjects, have a greater stroke volume response to incremental exercise. The increased stroke volume response is a determining factor of the increased cardiac output in these subjects, relative to the age-matched, untrained controls. Schairer *et al.* (1992) have similarly observed a larger exercising stroke volume as a major factor influencing enhanced cardiac output in young trained athletes compared to their sedentary counterparts. In the Schairer study, the athletes' enhanced stroke volume was due to increased end-diastolic volume, whereas their sedentary subjects' end-diastolic volume did not change during exercise.

Trained older athletes in the present study possessed a high aerobic power (mean of 54 ml.kg⁻¹min⁻¹) and the stroke volume of the Runners was significantly higher than that of Controls. Similar to that observed in endurance-trained subjects, the possible mechanism underlying the augmented stroke volume of endurance-trained older Runners may also be increased venous return, due possibly to training-induced hypervolaemia and the muscle pump, bringing about a large and early increase in venous return (Rowell, 1986). The results of a recent longitudinal training study by Carroll and colleagues (1995) indicates that older males and females increase blood volume as a result of endurance training. In addition, Stevenson *et al.* (1994) have shown that expanded blood volume is a key factor underlying the higher $\dot{V}O_{2max}$ of older trained females compared to untrained females. Also, preliminary data (Stevenson *et al.*, 1994) indicates that in older trained males expanded blood volume underlies their enhanced exercise stroke volume. Although we did not measure blood volume in the present study, Convertino (1991) has demonstrated that enhanced blood volume is associated with low resting heart rate.

Runners in this investigation recorded a resting heart rate of 50 b.min⁻¹.

Consequently, it is feasible that training-induced hypervolemia and increased venous return may be important factors determining the large increased stroke volume of older endurance trained Cyclists and Runners observed in this study.

5.1.2 Peak stroke volume

In contrast to previous studies (Astrand *et al.*, 1964; Higgenbotham *et al.*, 1986; Poliner *et al.*, 1980; Sullivan *et al.*, 1991) where stroke volume dynamics have been described as a linear increase to a maximal value peaking at approximately 40-50% of $\dot{V}O_{2max}$, stroke volume in the present study peaked at less than 20% of $\dot{V}O_{2max}$ in all three groups. The equivocality of stroke volume response during the early phase of aerobic exercise may be attributable to the inherent methodological limitations of most stroke volume assessment techniques.

For example, Ehsani *et al.* (1991), during supine cycle ergometer exercise, demonstrated that previously sedentary men (64 ± 3 years), who underwent twelve months of intense endurance training, significantly increased end-diastolic volume and stroke volume at peak exercise. Left ventricular function was assessed with electrocardiographic-gated blood pool imaging at steady state exercise. This technique measured stroke volume during the last 2 minutes of a 3-minute step protocol. As a result, the frequency of stroke volume sampling was limited and a detailed stroke volume response, including the initiation of exercise, was not assessed. Also, Ogawa *et al.* (1992) reported that the highest stroke volumes in older trained (63 ± 4 years; 47.6 ± 4.3 ml.kg⁻¹min⁻¹) and untrained males (63 ± 3 years; 27.2 ± 5.1 ml.kg⁻¹min⁻¹) were achieved at 50% of $\dot{V}O_{2max}$, and that stroke volume decreased between 50% and 100% of maximal effort. However, cardiac

output, which was measured by acetylene rebreathing, was assessed in the last 30 seconds of each 4-6 minute exercise trial (i.e., 50% of $\dot{V}O_{2\max}$ and 75% of $\dot{V}O_{2\max}$), and 4-6 minutes of progressively intense exercise that elicited $\dot{V}O_{2\max}$ during the last minute. Thus, due to a lack of data at the initiation of exercise, stroke volume response during light exercise remained unexplored. Similarly, Fleg *et al.* (1994) using radionuclide ventriculography during upright cycle ergometry attributed the increased cardiac index of trained older athletes (62.8 ± 6.9 years; 50.1 ± 4.9 ml. $\text{kg}^{-1}\text{min}^{-1}$) at exhaustion to an augmented stroke volume index. Images were taken during the last 2.5 minutes of each 3-minute exercise stage. Therefore, due to discontinuous measurement procedures, a detailed picture of stroke volume response dynamics throughout exercise remains unclear.

In contrast to rebreathing and echocardiography techniques, the impedance cardiography technique used in this study permitted the assessment of stroke volume on a more frequent basis during non-steady state exercise. Consequently, these results support the notion that stroke volume peaks at less than 20% of $\dot{V}O_{2\max}$, and is in contrast to the majority of the literature that has indicated that stroke volume is a linear increase to a maximal value followed by a levelling off at approximately 40-50% of $\dot{V}O_{2\max}$.

5.1.3 Stroke volume dynamics of untrained Controls

The data displayed in Figures 4.2 and 4.3 indicate that untrained healthy older males increased stroke volume during the early stages of exercise and support the findings of Rodeheffer *et al.* (1984) who found an age-related increase in stroke volume in untrained young and older males. Rodeheffer and colleagues suggested that the failure of past studies to screen for cardiovascular disease may account for

the lack of stroke volume response during exercise. Rodeheffer also suggests that with increasing age there is a shift from catecholamine-mediated increase in heart rate and decreased end-systolic volume to a greater dependence on the Frank Starling mechanism (higher stroke volume). Thus, it appears that increased end-diastolic volume and cardiac dilatation maintain cardiac output during exercise in older healthy males. Moreover, Fleg *et al.* (1994b) demonstrated the different effect of beta-adrenergic blockade on maximum exercise haemodynamics in young and older males. The greater decrease in maximum heart rate in younger males was compensated for by an increased stroke volume that did not occur in the older men. These findings indicate that in untrained, healthy older men reduced beta-adrenergic responsiveness during exercise may be compensated for by an enhanced stroke volume.

Increased stroke volume appears to be an important factor influencing cardiac output in both trained and healthy untrained older males. In contrast, to previous studies, young sedentary controls have consistently failed to increase stroke volume during moderate exercise (Boutcher *et al.*, 1994; Schairer *et al.*, 1992). However, although older subjects in the current study were untrained, they engaged in regular physical activity (e.g., golf and walking) and were relatively lean. Consequently, together with a greater reliance on the Frank-Starling mechanism, a healthy lifestyle (e.g., exercise, diet) may also contribute to the increased exercise stroke volume in untrained but healthy older individuals. These data support the notion that enhanced stroke volume is a major factor determining increased cardiac output in both trained and healthy untrained older males. However, the magnitude of the stroke volume increase in the untrained was significantly less than the trained older males. The

higher stroke volumes of the trained may possibly be due to their greater blood volume that has been observed in trained older males. This greater blood volume may result in a higher venous return, increased central venous pressure, and increased stroke volume response to exercise (Rowell, 1986).

5.1.4 Exercise mode specificity in cardiac adaptations

A second aim of the study was to compare the stroke volume response of trained older Runners to that of trained older Cyclists during upright cycle ergometry. These data indicate that absolute $\dot{V}O_{2peak}$ of Cyclists and Runners were not significantly different (Table 1), however, stroke volume of Runners was significantly higher than that of the Cyclists during the initial stages of cycle ergometry and significantly higher than Controls throughout the exercise protocol (Figure 4.2). Also, resting heart rate of both Cyclists and Runners were not significantly different, however, both groups had significantly lower resting heart rate compared to that of Controls (Table 1). These findings concur with previous research (Kasch *et al.*, 1973; McArdle *et al.*, 1976; Mutton *et al.*, 1993; Wilmore *et al.*, 1980) with young athletes that demonstrate, that similar cardiac adaptations occur as a result of endurance training. McArdle *et al.* (1976) hypothesised that any large muscle exercise such as running produces a general adaptation that results in reduced submaximal exercise heart rates and an increased oxygen pulse. Improved oxygen pulse indicates that the amount of oxygen transported by the blood per heart beat is increased and reflects improvement in oxygen extraction and/or stroke volume. Furthermore, similar to McArdle *et al.* (1976), Loftin *et al.* (1988) hypothesised that as the amount of exercising muscle mass increases, the relative influence of stroke volume to the determination of $\dot{V}O_{2max}$ increases. As a result,

after endurance arm training, small increases were observed in cardiac output (6%) and $\dot{V}O_{2\max}$ (7%) during leg exercise with no change in oxygen extraction. The authors concluded that central circulatory adaptation after arm exercise is also evident during leg exercise. Thus, these studies provide indirect evidence supporting the current finding that endurance running results in enhanced stroke volume response to cycle ergometry in older males.

Additional indirect support for cardiac adaptations accruing through endurance training are provided by studies demonstrating improved exercise performance with untrained limbs (Lewis *et al.*, 1980; Thompson *et al.*, 1981). Thompson *et al.* (1981) demonstrated an increased left ventricular ejection time with exercise training. Left ventricular ejection time correlates negatively with heart rate and positively with stroke volume. Thus, the increase in left ventricular ejection time observed by Thompson *et al.* (1991) indicates that stroke volume contributes to improved exercise capacity in untrained limbs. The current study also demonstrated significantly lower resting heart rate and increased left ventricular ejection time in the trained subjects at rest compared to controls. These data indicate that cardiac adaptations such as lower heart rate and increased stroke volume during submaximal exercise accruing as a result of endurance running training also enhance cardiac performance during cycle exercise. Thus, while such adaptations appear to be exercise mode specific (Runners greater exercising stroke volume than Cyclists), there is a transference of the benefits of this adaptation to other exercise modes. The incongruity of the Cyclists' higher relative $\dot{V}O_{2peak}$ (Table 1) and the Runners' higher stroke volume (Figure 4.2), is an interesting result of this study. Runners, with an average respiratory exchange ratio of 1.0, failed to fulfill the criteria for

$\dot{V}O_{2max}$. As a result Controls (respiratory exchange ratio of .96) and Runners peaked rather than reaching true maximal oxygen uptake. Local muscle fatigue may have limited exercise performance in Runners and Controls; in contrast, the Cyclists recorded a respiratory exchange ratio of 1.12 that fulfilled the criteria for $\dot{V}O_{2max}$. As mentioned previously, the stroke volume difference between endurance trained Cyclists and untrained controls may be due to training-induced hypervolaemia. Similarly, the increased blood volume accruing through run-training may be the basis for the enhanced stroke volume performance during cycle ergometry (Convertino *et al.*, 1991).

Thus, overall, Runners' bike exercise performance compared to Cyclists may have been limited by local muscle fatigue. However, Runners' demonstrated a higher stroke volume response, thus supporting the notion that cardiac adaptations (such as increased stroke volume) as a result of endurance running exercise transfers to enhanced stroke volume response to cycle exercise.

5.1.5 Cardiac output and total peripheral resistance

Runners and Cyclists compared to Controls performed double the workload at each exercise heart rate (Figure 4.1). The Runners and Cyclists also displayed the largest increase in peak cardiac output (Figure 4.4) and the greatest decline in total peripheral resistance (Figure 4.6) from rest to peak effort. The finding that the total peripheral resistance of the Trained was significantly lower than that of the Controls during exercise is consistent with previous research (Fleg *et al.*, 1994a; Martin *et al.*, 1991). A potential vascular response to training-induced hypervolaemia, and increased cardiac output during exercise, may be the release of nitric oxide from endothelial cells occurring as a result of increased blood flow. This may result in

vasodilation of arteries and arterioles (Cooke *et al.*, 1990). Also the ability of aerobic-training to influence vasculature dilation is reinforced by a recent investigation (Haskell *et al.*, 1993). The results of Haskell's study indicate that middle-aged endurance runners have a significantly greater arterial dilating capacity compared to sedentary controls. After an infusion of nitroglycerin, which is an endothelium-independent coronary vasodilator, the runners total coronary cross-sectional area was double that of the untrained, thus reflecting a greater vasodilatory capacity. Further evidence of a potential vascular response to endurance exercise is provided by Vaitkevicius *et al.* (1993), who showed that aerobic exercise may reduce the stiffening of the arterial tree that is associated with aging. Thus, in the current study Cyclists and Runners displayed the largest increase in peak cardiac output and the greatest decline in total peripheral resistance during exercise.

Ageing is associated with reduced endothelium-dependent vasodilation and decreased peripheral vasodilatory capacity that may contribute to the pathogenesis of cardiovascular disease and hypertension (Taddei *et al.*, 1995). Importantly, results of this study and others (Hagberg *et al.*, 1985; Martin *et al.*, 1987) indicate that aerobic training enhances vasodilatory capacity and reduces total peripheral resistance during exercise (Figure 4.6). Overall, data from the current study indicate that trained older Cyclists and Runners were able to significantly lower total peripheral resistance (decrease afterload) during exercise compared to untrained controls.

5.1.6 Cardiovascular variables at rest

At rest trained Runners (50 b.min⁻¹) and Cyclists (56 b.min⁻¹) demonstrated significantly lower resting heart rate compared to untrained Controls (67 b.min⁻¹).

These data concur with previously observed training-induced decrease in resting heart rate in older athletes (Hagberg *et al.*, 1985; Heath *et al.*, 1981; Fleg *et al.*, 1994). Consistent with previous research the Runners displayed a larger stroke volume and stroke index than Controls at rest (Fleg *et al.*, 1994). This enhanced resting stroke volume may be due to training-induced hypervolaemia because of the higher training volume and intensity. In addition, the Runners' may have a part compensatory effect as evidenced by the low resting heart beat of $50 \text{ b} \cdot \text{min}^{-1}$ which may be considered as a possible marker of enhanced blood volume (Convertino, 1991). However, the interaction between hypervolaemia and the shift in control of heart function are unclear. Kanstrup *et al.* (1982) suggested that a change in autonomic factors cannot be ruled out and that exercise bradycardia associated with endurance training may be due to a depressed sympathetic stimulation after blood volume expansion. Thus, the Runners' low resting heart rate may reflect the influence of endurance training upon the autonomic function of the heart by increasing parasympathetic influence upon the heart (Kenny, 1985). Significantly, the influence of aerobic exercise on parasympathetic control of the heart has major consequences for the ageing heart because previous research indicates that lack of parasympathetic tone is associated with ageing and negative cardiovascular health (Adgey *et al.*, 1982; Eckberg, 1980; Hinkle *et al.*, 1972; Kent *et al.*, 1973). Moreover, the lower resting heart rate of the endurance trained Runners is efficacious from a cardiovascular health perspective, as rate pressure product (systolic blood pressure x heart rate) which is linear to myocardial oxygen consumption is also lower (Lehman & Keul, 1986).

At rest, the rate pressure product of untrained Controls was higher than that

of Cyclists and Runners. Rate pressure product is positively correlated with coronary blood flow and myocardial oxygen consumption in healthy subjects over a wide range of exercise intensities (Kitamura *et al.*, 1972; Nelson *et al.*, 1972).

Previous research indicates that aerobic training of specific duration and intensity results in higher rate pressure product being achieved before the appearance of ischemic symptoms (Rogers *et al.*, 1987). Consequently in this study the data may provide indirect evidence that untrained Controls compared to trained Cyclists and trained Runners have reduced levels of myocardial oxygenation at rest, possibly due to reduced coronary vascularisation or arterial obstruction.

Runners and Cyclists at rest had significantly longer left-ventricular ejection time compared to Controls which supports previous research indicating increased left-ventricular ejection time after training (Thompson *et al.*, 1981). Significantly, cardiac disease is characterised by a decline in left-ventricular performance with distinctive changes in systolic time intervals. For example, reduced left-ventricular ejection time is positively correlated with reduced stroke volume which may in turn be associated with cardiac disease (Weissler, 1977). In contrast, trained Runners and Cyclists in this study demonstrated enhanced left-ventricular performance characterised by increased stroke volume and longer left-ventricular ejection time.

5.2 CONCLUSIONS

Overall, all three groups showed a significant increase in stroke volume and a significant decline in total peripheral resistance. During exercise, however, Cyclists and Runners displayed greater stroke volume than Controls which was one of the major contributing factors for their greater $\dot{V}O_{2peak}$. Also, stroke volume peaked at less than 20% of $\dot{V}O_{2peak}$ in both the trained and untrained older subjects. This early

increase in stroke volume is a determining factor of the increased cardiac output during exercise not only in the trained but also in the untrained, healthy Controls. Overall the Cyclists displayed the greatest $\dot{V}O_{2peak}$ although the Runners had a larger stroke volume as a determining factor of their $\dot{V}O_{2peak}$. This finding indicates that in older males, endurance exercise (e.g., running) results in enhanced stroke volume response to cycle exercise. Furthermore, the data provides additional support for the concept of aerobic exercise intervention for the older segment of the population that may enhance their quality of life through improved cardiovascular health.

5.3 LIMITATIONS

The problem of cross-sectional design, which isolates sections of life as opposed to a full life span in a longitudinal study, is difficult to circumvent. However, some subjects indicated that their lifestyle had changed dramatically at various stages of their life due to increased health awareness and/or retirement. For example, greater exercise participation, ceased smoking, diet and weight control, and in particular moderate consumption or abstinence from alcohol were incorporated into lifestyle. Consequently, isolating the impact of age and lifestyle factors such as exercise on cardiac function may still be confounded in a longitudinal study.

Because the sample population were screened they may not have been representative of the general population. However, it was important from an ethics perspective to identify subjects with cardiovascular disease and/or cardiac abnormality who may have been a potential risk during incremental exercise testing. Also, in previous studies with older males where stroke volume was found to increase subjects had been screened for cardiovascular disease. In addition, because

subjects were recruited from veteran cycle and running clubs they were more likely to be highly motivated, enthusiastic, and interested in their health compared to the average 65 year old. Elite masters athletes are a unique group and in some cases may represent extremes of activity patterns which are more typical of young elite competitors. Thus, these results may only be generalised to the trained and healthy older male members of the community.

The measurement of $\dot{V}O_{2\max}$ is problematical with older subjects who are unfamiliar with intense exercise and experience difficulty in achieving a true plateau. Thus, the respiratory exchange ratio (.96) of Controls in this study indicates that the problem is exaggerated with older subjects because of a lack of familiarity with exercise testing and increased physiological differences among older individuals.

5.4 RECOMMENDATIONS FOR FUTURE RESEARCH

Future research could focus on further delineating the mechanisms underlying the larger stroke volume response of the trained older athletes by also incorporating blood volume measurements. These blood volume measurements, prior to, and at the completion of a longitudinal training study may help clarify the factors underlying the stroke volume differences between trained and untrained older subjects.

How the trained older athletes increase venous return so early on in exercise is unclear. For instance, the ability of the exercise muscle pumps to effectively return blood to the heart together with increased central venous pressure via increased blood volume may be the basis of the large, early increase in stroke volume. Further research isolating these mechanical aspects of the enhanced stroke volume of the trained elderly is warranted.

CHAPTER SIX: REFERENCE LIST

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APPENDICES

Appendix A Human experimentation ethics approval



Office of the Vice-Principal (Administration)

Academic & Student Services Branch

CONDITIONAL APPROVAL

In reply please quote: DC:KM HE 94/23

Further Information: Karen McRae (Ext 4457)

30 March 1994

Mr Peter McLaren
Biomedical Sciences
University of Wollongong
Northfields Avenue
Wollongong NSW 2522

Dear Mr McLaren,

I am pleased to advise that the following Human Research Ethics application has been conditionally approved:

Ethics Number: HE 94/023

Project Title: Cardiac response of elderly trained cyclists and runners, and untrained males during bicycle ergometry.

Name of Researchers: Mr Peter McLaren

Approval Date: 22 March 1994

Duration of Clearance: 21 March 1995

This approval is granted subject to satisfactory clarification of the following matters:

- (i) Information in the Information for Subjects be clarified for the potential subjects.
- (ii) Participants free to "refuse consent" rather than "deny consent" in Item Six.
- (iii) A physician familiar with resuscitation techniques be present: this is not clear in the protocol but it is referred to elsewhere.

- (iv) that the Consent Form be amended to note that any complaints regarding the conduct of the research may be directed to the Secretary of the University of Wollongong Human Research Ethics Committee (telephone 214457).

please provide written evidence that this condition has been satisfied to the Secretary of the Committee before the commencement of your research, or approval will be withdrawn.

This certificate relates to the research protocol submitted in your application of 10 March 1994. It will be necessary to inform the Committee of any changes to the research protocol and seek clearance in such an event.

Please note that experiments of long duration must be reviewed annually by the Committee and it will be necessary for you to apply for renewal of this application if experimentation is to continue beyond one year.



Chairperson
Human Research Ethics Committee

Head, Department of Biomedical Science

Appendix B Informed consent.

SUBJECT INFORMED CONSENT

The researchers conducting this project support the principles governing both the ethical conduct of research, and the protection at all times of the interests, comfort, and safety of subjects.

This form and the accompanying "Information for Subjects" leaflet are given to you so that you may be fully informed of the experimental procedures and possible risks that accompany participation in this study.

Your signature below indicates six things:

- (1) you have received the "Information for Subjects" leaflet;
- (2) you have read its contents;
- (3) you have been given the opportunity to discuss the contents with one of the researchers prior to commencing the experiment;
- (4) you clearly understand the procedures and possible risks of participation in the study;
- (5) you voluntarily agree to participate in the project; and
- (6) your participation may be terminated at any point in time without jeopardising in any way your involvement with the University of Wollongong.

Any concerns, complaints, or further questions may be directed to Dr Steve Boutcher (Department of Biomedical Science: phone 214093). Subsequent inquiries may be directed to the Secretary of the University of Wollongong Human Research Ethics Committee, phone 214457).

Signed: _____ Date: _____

Appendix C Personal health and exercise history questionnaire.

PERSONAL HEALTH HISTORY

Please complete this form as accurately as possible.

Name: _____ Date: _____ Age: _____

Height: _____ Weight: _____ Sex: _____

Date of birth _____ Ethnicity: _____

1. GENERAL MEDICAL HISTORY

circle one

Any medical complaints? (Please specify) YES NO

Are you on any medication? YES NO

Specify _____

Adrenal disease? YES NO

Fainting spells? YES NO

Hypoglycemia? YES NO

Seizures? YES NO

Diabetes? YES NO

Kidney problems? YES NO

Stomach ulcers? YES NO

2. EXERCISE HISTORY

Do you jog, cycle or swim? YES NO

If yes then specify:

a. how many times a week? _____

b. how long is each session? _____

c. what kind of pace? _____

d. how long have you been regularly exercising?

e. how many years overall have you been exercising? _____

Do you do any other form of exercise?

Specify _____

a. how many times a week? _____

b. how long each session? _____

c. how many years have you participated in this activity? _____

3. CARDIORESPIRATORY HISTORY

Any heart disease now? YES NO

Any heart disease in past? YES NO

Heart murmurs? YES NO

Occasional chest pains? YES NO

Fainting? YES NO

Asthma or allergies? YES NO

Family history of heart disease? YES NO

High blood pressure? YES NO

Shortness of breath after walking YES NO

flights of stairs?

4. MUSCULAR HISTORY

Any muscle injuries now?	YES	NO
Muscle injuries in past?	YES	NO
Muscle pains during exercise?	YES	NO
Family history of muscle pains?	YES	NO

5. BONE-JOINT HISTORY

Any bone or joint injuries now?	YES	NO
Any in past?	YES	NO
Ever had swollen joints?	YES	NO

6. PREPAREDNESS FOR TESTING

SPECIFY

a. Any food during the last 8 hours?	YES	NO
b. Any liquids during the last 8 hours? (except water).	YES	NO
c. Any caffeine during the last 8 hours?	YES	NO
d. Any medication during the last 8 hours? (including insulin, except for basal infusion in pump patients).	YES	NO
e. Any over the counter drugs during the last 8 hours? (aspirin, antihistamines, nasal sprays, etc).	YES	NO
f. Any alcohol in last 24 hours?	YES	NO
g. Any tobacco during the last 8 hours?	YES	NO
h. Any vigorous exercise in the last 24 hours? (any exercise not part of patient's daily routine ie., routine jogging ok, but marathon running is not. No exercise morning of test).	YES	NO
i. Any emotional upset in last 24 hours? Depression, crying episodes, anxiety from personal trauma (death, divorce, car accident dentist, etc).	YES	NO
j. Acute illness in last 48 hours? (cold, flu, measles, etc).	YES	NO

Appendix D Resting and exercise means and standard deviations for all cardiovascular variables of trained older Cyclists, Runners, and untrained Controls.

Table D-1: Supine and seated resting cardiac output ($L \cdot min^{-1}$). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	5.26 (.24)	5.41 (.40)	5.86 (.24)
	2	5.19 (.27)	5.42 (.42)	5.81 (.29)
Seated rest	1	4.08 (.27)	4.21 (.34)	4.25 (.28)
	2	3.86 (.34)	4.30 (.37)	4.24 (.29)
Cycle ergometry	1	8.29 (.39)	10.21 (.59)	7.40 (.48)
	2	9.76 (.42)	11.15 (.62)	8.32 (.47)
	3	11.03 (.54)	12.12 (.75)	9.47 (.38)
	4	12.00 (.49)	13.38 (.90)	10.78 (.47)
	5	13.02 (.38)	14.74 (.90)	11.78 (.55)
	6	13.85 (.55)	15.37 (1.01)	12.67 (.64)
	7	14.81 (.46)	16.33 (.99)	13.27 (.71)

Note: During each rest period, data were sampled at two separate points during the steady state. However, during exercise, data were sampled at heart rates of 90, 100, 110, 120, 130, 140, and 150 $b \cdot min^{-1}$.

Table D-2: Supine and seated resting stroke volume (ml). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	93.7 (3.8)	105.9 (5.8)	87.9 (4.5)
	2	92.4 (4.0)	106.4 (6.1)	87.1 (4.5)
Seated rest	1	65.0 (2.8)	75.8 (5.1)	62.1 (3.9)
	2	63.7 (4.2)	76.2 (5.3)	61.7 (4.0)
Cycle ergometry	1	89.9 (3.6)	112.5 (6.2)	80.8 (5.4)
	2	96.4 (3.9)	111.8 (6.3)	83.8 (4.9)
	3	98.2 (4.7)	110.0 (6.7)	85.7 (3.5)
	4	98.6 (4.2)	111.2 (7.1)	89.7 (4.5)
	5	98.8 (2.8)	113.5 (6.6)	90.5 (4.5)
	6	98.3 (4.0)	112.9 (7.3)	91.1 (4.9)
	7	99.7 (3.3)	115.2 (6.4)	89.6 (5.3)

Note: During each rest period, data were sampled at two separate points during the steady state. However, during exercise, data were sampled at heart rates of 90, 100, 110, 120, 130, 140, and 150 b.min⁻¹.

Table D-3: Supine and seated resting stroke index (ml/m²). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	50.7 (2.0)	55.4 (3.1)	44.9 (2.3)
	2	50.0 (2.0)	55.6 (3.3)	44.5 (2.3)
Seated rest	1	35.4 (2.1)	39.7 (2.6)	31.7 (2.1)
	2	34.9 (2.9)	39.9 (2.8)	31.6 (2.2)
Cycle ergometry	1	49.0 (2.6)	58.6 (2.7)	41.3 (2.7)
	2	52.5 (2.7)	58.3 (2.9)	42.8 (2.4)
	3	53.4 (2.9)	57.3 (2.9)	43.8 (1.8)
	4	53.5 (2.4)	57.9 (3.2)	45.7 (2.1)
	5	53.5 (1.4)	59.1 (2.8)	46.2 (2.3)
	6	53.2 (2.0)	58.8 (3.2)	46.5 (2.5)
	7	54.0 (1.7)	60.1 (3.0)	45.7 (2.7)

Note: During each rest period, data were sampled at two separate points during the steady state. However, during exercise, data were sampled at heart rates of 90, 100, 110, 120, 130, 140, and 150 b.min⁻¹.

Table D-4: Supine and seated resting heart rate (b.min⁻¹). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	56.2 (1.2)	50.9 (2.5)	67.6 (3.4)
	2	56.0 (1.2)	50.8 (2.7)	67.7 (3.4)
Seated rest	1	62.5 (2.5)	55.8 (3.3)	69.3 (3.9)
	2	60.1 (2.0)	56.6 (3.8)	69.7 (4.3)
Cycle ergometry	1	92.0 (0.9)	90.7 (1.0)	91.6 (0.7)
	2	101.1 (0.9)	99.8 (1.0)	99.3 (1.0)
	3	112.3 (0.5)	110.1 (0.9)	110.5 (0.9)
	4	121.8 (0.9)	120.0 (1.5)	120.4 (1.2)
	5	131.8 (0.6)	129.7 (1.4)	130.2 (1.0)
	6	140.9 (0.4)	136.4 (2.7)	139.4 (1.5)
	7	148.7 (1.4)	142.0 (3.8)	147.6 (2.4)

Note: During each rest period, data were sampled at two separate points during the steady state. However, during exercise, data were sampled at heart rates of 90, 100, 110, 120, 130, 140, and 150 b.min⁻¹.

Table D-5: Supine and seated resting rate pressure product. Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	87.3 (3.0)	75.1 (4.1)	106.0 (8.1)
	2	91.4 (3.5)	76.5 (4.0)	104.4 (6.7)
Seated rest	1	104.1 (7.0)	89.7 (5.6)	113.3 (9.5)
	2	101.8 (4.7)	91.1 (6.5)	115.7 (9.5)
Cycle ergometry	1	191.8 (8.2)	172.2 (4.9)	170.6 (7.0)
	2	217.4 (8.1)	204.1 (5.4)	196.6 (7.9)
	3	255.3 (7.5)	233.8 (5.3)	224.8 (9.0)
	4	286.7 (8.8)	260.7 (7.6)	256.9 (9.0)
	5	318.1 (9.8)	294.5 (7.8)	283.1 (7.6)
	6	353.2 (9.5)	314.0 (10.4)	311.8 (8.6)
	7	375.1 (8.9)	326.2 (12.9)	335.3 (8.3)

Note: During each rest period, data were sampled at two separate points during the steady state. However, during exercise, data were sampled at heart rates of 90, 100, 110, 120, 130, 140, and 150 b.min⁻¹.

Table D-6: Supine and seated resting total peripheral resistance. Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	1685.8 (114.3)	1575.0 (165.9)	1491.9 (93.9)
	2	1797.6 (124.4)	1627.9 (182.5)	1491.2 (68.3)
Seated rest	1	2430.2 (200.8)	2164.9 (173.4)	2292.6 (197.2)
	2	2659.2 (273.6)	2168.1 (222.1)	2348.2 (212.5)
Cycle ergometry	1	1320.0 (73.2)	986.6 (55.6)	1407.0 (69.5)
	2	1115.7 (51.5)	966.1 (47.9)	1315.1 (57.9)
	3	1029.0 (53.8)	900.9 (49.9)	1165.0 (59.8)
	4	978.5 (45.6)	824.7 (49.9)	1078.4 (46.1)
	5	913.6 (30.2)	764.9 (47.1)	1013.3 (48.5)
	6	882.6 (37.5)	757.2 (42.2)	968.9 (55.8)
	7	810.4 (30.2)	681.3 (39.4)	936.9 (52.4)

Note: During each rest period, data were sampled at two separate points during the steady state. However, during exercise, data were sampled at heart rates of 90, 100, 110, 120, 130, 140, and 150 b.min⁻¹.

Table D-7: Supine and seated resting left-ventricular ejection time. Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	368.2 (13.1)	355.2 (16.1)	309.6 (12.1)
	2	370.6 (11.2)	351.4 (15.1)	305.6 (12.2)
Seated rest	1	305.8 (16.4)	298.1 (17.2)	284.0 (15.9)
	2	289.8 (12.2)	299.4 (17.4)	276.4 (14.9)
Cycle ergometry	1	291.8 (14.4)	290.5 (14.7)	278.0 (10.7)
	2	282.8 (14.0)	272.3 (13.3)	273.0 (13.2)
	3	257.6 (10.1)	254.5 (11.5)	255.6 (10.3)
	4	250.4 (6.8)	240.0 (10.3)	246.4 (9.4)
	5	237.0 (5.5)	233.0 (11.1)	236.8 (7.4)
	6	227.6 (6.5)	221.0 (12.2)	224.4 (7.3)
	7	235.0 (6.7)	223.0 (13.8)	208.4 (6.7)

Note: During each rest period, data were sampled at two separate points during the steady state. However, during exercise, data were sampled at heart rates of 90, 100, 110, 120, 130, 140, and 150 b.min⁻¹.

Table D-8: Supine and seated resting cardiac index (L/min/m²). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	2.8 (0.1)	2.8 (0.2)	3.0 (0.1)
	2	2.8 (0.1)	2.8 (0.20)	2.9 (0.1)
Seated rest	1	2.2 (0.1)	2.1 (0.1)	2.1 (0.1)
	2	2.1 (0.2)	2.2 (0.1)	2.1 (0.1)
Cycle ergometry	1	4.5 (0.2)	5.3 (0.2)	3.7 (0.2)
	2	5.3 (0.2)	5.8 (0.20)	4.2 (0.2)
	3	6.0 (0.3)	6.3 (0.3)	4.8 (0.2)
	4	6.5 (0.2)	6.9 (0.4)	5.5 (0.2)
	5	7.0 (0.2)	7.6 (0.3)	6.0 (0.3)
	6	7.5 (0.2)	7.9 (0.4)	6.4 (0.3)
	7	8.0 (0.2)	8.5 (0.4)	6.7 (0.3)

Note: During each rest period, data were sampled at two separate points during the steady state. However, during exercise, data were sampled at heart rates of 90, 100, 110, 120, 130, 140, and 150 b.min⁻¹.

Table D-9: Supine and seated resting systolic blood pressure. Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	156.0 (5.9)	147.6 (3.9)	156.5 (8.1)
	2	163.2 (5.6)	151.2 (4.3)	154.4 (6.3)
Seated rest	1	167.3 (10.8)	161.4 (5.0)	162.6 (8.8)
	2	170.9 (9.3)	161.32 (5.0)	165.4 (8.1)
Cycle ergometry	1	208.1 (7.8)	190.0 (5.7)	186.1 (7.4)
	2	214.8 (7.1)	204.4 (4.9)	197.9 (7.5)
	3	227.3 (6.6)	212.1 (3.8)	203.3 (7.8)
	4	235.3 (6.6)	217.0 (5.1)	213.3 (7.2)
	5	241.2 (6.8)	226.8 (4.5)	217.5 (6.0)
	6	250.6 (6.8)	230.0 (5.0)	223.9 (6.6)
	7	253.3 (6.1)	229.3 (5.0)	227.5 (6.1)

Note: During each rest period, data were sampled at two separate points during the steady state. However, during exercise, data were sampled at heart rates of 90, 100, 110, 120, 130, 140, and 150 b.min⁻¹.

Table D-10: Supine and seated resting diastolic blood pressure. Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	84.7 (4.6)	75.0 (3.1)	83.0 (4.3)
	2	88.9 (4.9)	76.8 (2.8)	83.3 (3.8)
Seated rest	1	96.3 (7.6)	80.2 (4.5)	92.7 (4.1)
	2	94.9 (8.7)	79.8 (5.1)	95.0 (3.9)
Cycle ergometry	1	97.4 (4.5)	88.3 (3.3)	97.8 (4.2)
	2	94.3 (4.4)	96.0 (9.0)	102.5 (3.8)
	3	95.4 (4.8)	92.2 (3.6)	100.8 (5.5)
	4	99.6 (4.1)	90.5 (2.8)	104.1 (5.9)
	5	101.5 (5.4)	90.7 (3.7)	104.3 (5.4)
	6	101.5 (4.9)	96.1 (4.2)	104.3 (5.7)
	7	97.4 (4.5)	88.6 (7.7)	100.8 (5.4)

Note: During each rest period, data were sampled at two separate points during the steady state. However, during exercise, data were sampled at heart rates of 90, 100, 110, 120, 130, 140, and 150 b.min⁻¹.

Table D-11: Supine and seated resting mean arterial blood pressure. Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Supine rest	1	108.2 (4.8)	99.0 (2.9)	107.2 (4.9)
	2	113.2 (5.0)	101.1 (2.5)	106.6 (4.1)
Seated rest	1	119.7 (8.3)	107.0 (3.1)	115.5 (3.5)
	2	120.0 (8.6)	106.5 (3.1)	118.1 (4.2)
Cycle ergometry	1	133.8 (4.8)	122.0 (2.4)	126.9 (4.3)
	2	134.2 (4.9)	131.8 (5.8)	133.8 (4.0)
	3	139.1 (4.6)	131.9 (2.8)	134.6 (4.7)
	4	144.4 (3.7)	132.2 (2.4)	140.1 (4.7)
	5	147.8 (4.4)	135.7 (3.0)	141.9 (3.7)
	6	151.0 (4.2)	140.4 (3.6)	143.7 (4.5)
	7	148.8 (4.1)	135.1 (5.6)	143.3 (3.9)

Note: During each rest period, data were sampled at two separate points during the steady state. However, during exercise, data were sampled at heart rates of 90, 100, 110, 120, 130, 140, and 150 b.min⁻¹.

Table D-12: Exercise work rates (Watts). Data are means with standard errors of the means in parenthesis.

Activity	Data	Cyclists	Runners	Controls
Cycle ergometry	1	62.0 (5.7)	59.0 (9.3)	29.0 (3.7)
	2	90.0 (6.3)	96.3 (11.6)	50.0 (4.4)
	3	123.0 (4.9)	125.4 (10.9)	69.0 (6.9)
	4	151.0 (7.0)	155.4 (12.0)	90.0 (7.3)
	5	176.0 (6.5)	180.0 (12.2)	108.0 (6.2)
	6	203.0 (7.8)	199.0 (11.4)	123.0 (5.1)
	7	228.0 (9.6)	221.8 (10.9)	137.0 (4.2)

Note: Exercise workrate (Watts) data were sampled at heart rates of 90, 100, 110, 120, 130, 140, and 150 b.min⁻¹.

Appendix E Exercise questionnaire

Q1. What is your predominant mode of exercise?

List any supplementary modes of exercises.

Q2. How many exercise sessions in your main mode would you average per week?

Minimum? Maximum?

Similarly how many sessions in other modes? Minimum? Maximum?

Q3. Are the above patterns of exercise all year round or do they fluctate

eg., seasonal, competitive phase, how you feel;

Q4. What is the average duration of your main mode exercise session?

Minimum Maximum

Q5. How many years have you been exercising regularly?

Q6. Do you participate in any competitive events?

If so indicate participation during the last 12 months;

Q7. Do you exercise at any particular intensity ? For example, by monitoring heart rate or rest interval?

Do you monitor your resting pulse? If so, when, and what would your resting pulse normally be?

Do you know your maximum heart rate?

Q8. Do you quantify your training load? For example, record distance.

Could you outline your personal best times and/or achievements

with particular detail for the last 12 months

(please complete on back of page)

Appendix F Information for subjects

INFORMATION FOR SUBJECTS

ITEM 1: PROJECT OBJECTIVES

The aim of the project is to investigate the cardiovascular response of trained and untrained elderly males during cycle ergometry.

ITEM 2: RATIONALE

Aerobically trained elderly individuals have a different cardiovascular response compared to their untrained counterparts to the stress of exercise. Typically trained elderly individuals who participate regularly in aerobic exercise have an enhanced oxygen uptake capacity due to an augmented stroke volume and arterio-venous difference. The mechanisms underlying the increased stroke volume of the trained elderly have not been identified.

ITEM 3: TEST PROCEDURES

Measures of cardiovascular function, such as heart rate, stroke volume, contractility, and blood pressure, will be collected during the experimental session. Testing will involve application of surface electrodes and blood pressure cuff on the arm. The experiment consists of an initial baseline session where resting heart function will be assessed. During a separate session subjects will be required to pedal a bicycle ergometer at maximum effort.

ITEM 4: RISKS AND DISCOMFORTS

During the initial session, your cardiac function will be screened for gross abnormalities by a cardiac nurse. During the second experimental session it is anticipated that your heart rate and blood pressure will rise, to high levels towards the end of the stress test. Cycle ergometry will also induce leg muscle fatigue; During the exercise session you will be supervised by an experienced cardiac nurse (from the Port Kembla Hospital cardiac rehabilitation centre) and a physician familiar with resuscitation techniques.

ITEM 5: INQUIRIES

Questions concerning the procedures and/or rationale used in this study are welcome at any time. Please ask for clarification of any point which you feel is not explained to your satisfaction. Your initial contact person is the investigator conducting this project (Dr Steve Boucher, Department of Biomedical Science: phone 214093). Subsequent inquiries may be directed to the Secretary of the University of Wollongong Human Research Ethics Committee, phone 214457).

ITEM 6: FREEDOM OF CONSENT

Participation in this project is entirely voluntary. You are free to refuse consent before or during the experiment. In the latter case such withdrawal of consent should be made at the time you specify, and not at the end of a particular trial. Your participation and/or withdrawal of consent will not influence your present and/or future involvement with the University of Wollongong. You have the right to withdraw from any experiment, and this right shall be preserved over and above the goals of the experiment.

ITEM 7: CONFIDENTIALITY

All questions, answers, and results of this study will be treated with absolute confidentiality. Subjects will be identified in the resultant manuscripts, reports or publications by the use of subject codes only.